

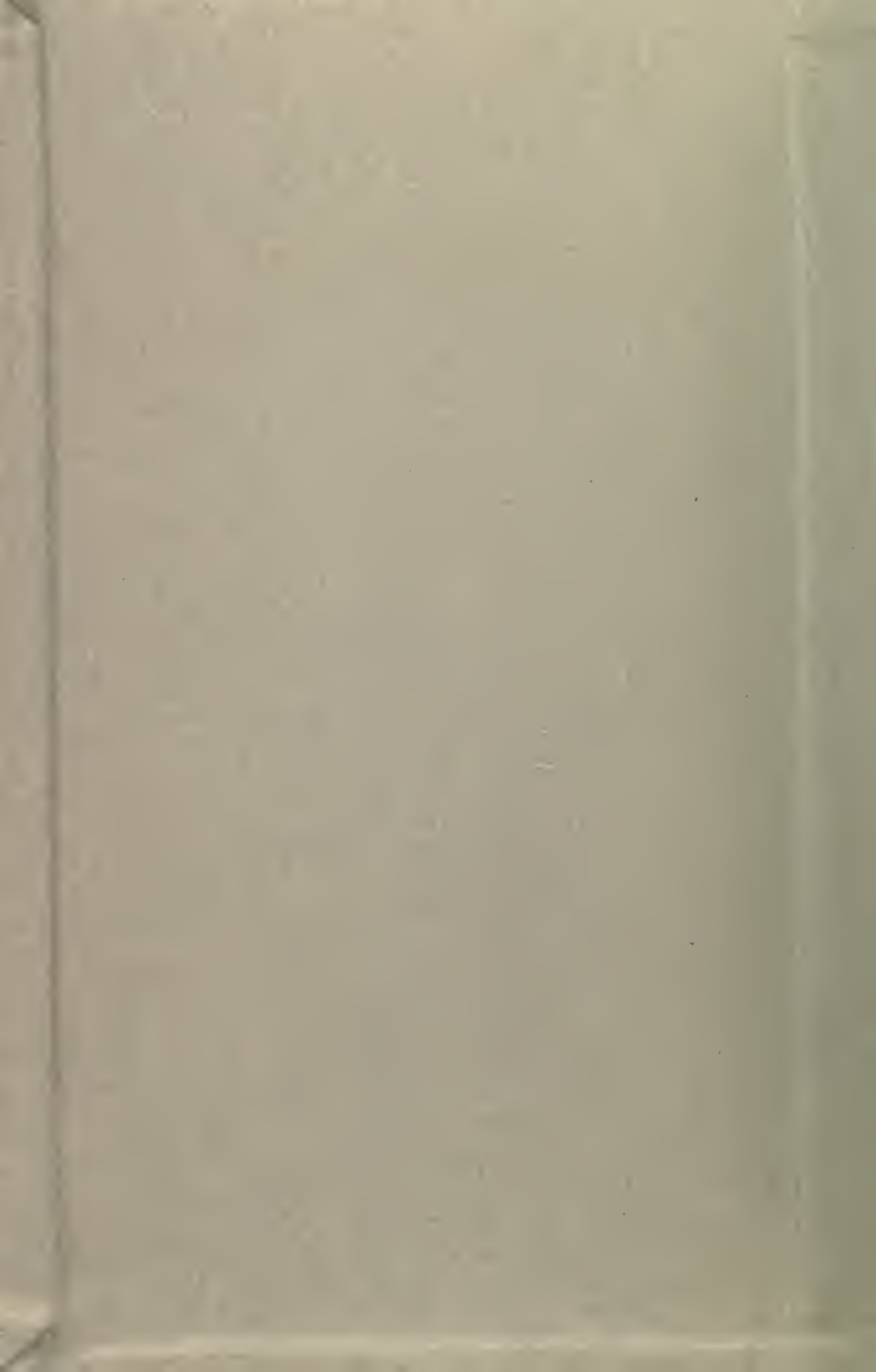
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The
ESSENTIALS
of
Clinical Toxicology

by

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Los Angeles, Cal.

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PREFACE

The publication of this discussion of poisoning and its treatment was prompted by the difficulty of finding among the various manuals on the subject any satisfactory presentation of its problems from the exclusive standpoint of the physician engaged in general practice, who, from force of circumstances, sees so few cases of acute poisoning that he must, in his management of them, rely not upon set procedures made familiar by frequent use but upon the application of a few more general principles. Therefore, in this presentation, the effort is made to relate the essential phenomena of poisoning with the more familiar facts of pharmacology, pathology, and general therapeutics, to serve particularly the needs of the beginning student of the subject.

Altho some of the time-honored procedures in the management of poisoning have either been omitted or cited as inferior to certain non-medicinal measures, this has been done only in instances where there is found in the literature ample justification for it, and where the rationale of the newer procedure is acceptable. Credit for the various ideas expressed cannot be specifically given to the authors consulted. Among these, however, are the following: Toxicology—Witthaus, Emerson, Reese, Brundage, Tanner, Autenrieth, Rambousek; Handbook of Therapy of A.M.A.; Willcox in "A System of Treatment," edited by Latham; Index of Differential Diagnosis; A. R. Short, in International Medical Annual, 1915; Bastedo, *Materia Medica*; Hydrotherapy, Abbott; Tyrode, Pharmacology; Greene, Pharmacology; Potter, *Materia Medica*; Medical Chemistry and Toxicology, Holland; Census Reports; Practice of Medicine, Osler, Tyson; Shock, in "Circulation in Health and Disease," Wiggers; and others.

L. C. C.

January, 1916.

INTRODUCTION

The term Clinical Toxicology is used to designate those classified items of our knowledge concerning poisons, their common sources, their effects upon the human body, and the methods to be used in assisting the body to recover from their effects, which are needed by the physician in managing cases of poisoning. An exact definition of the word "poison" is difficult to give, because of the great variety of substances which may, directly or indirectly, cause death by their chemical activities. Excepting when precision of diction is demanded, we may state, that a poison is any substance which, acting chemically in the body or upon it, tends to produce serious injury or death. The following are more satisfactory when exactness is desired:

A true poison is a substance possessing such a quality and degree of chemical activity that, in quantities readily absorbed, when present in or acting upon the blood, it tends uniformly to cause death or serious bodily harm.

A corrosive poison is a substance capable of causing death or injury by its chemical action upon a tissue with which it comes in direct contact.

It is a thoughtless notion of the physician's duties in the realm of toxicology, which limits his activities to the vigorous application of drastic measures during the time when a victim of poisoning is dangerously affected. Meeting the emergency is only the beginning of his duty. Following, must come his painstaking care of the patient to bring him back to the highest attainable degree of health, and to avoid the serious sequelæ in so far as possible. Even this statement omits reference to the physician's duty, in general practice, to detect early signs of the various chronic poisonings where only obscure illness is suspected by the patient.

In practice, instances of the poisonous actions of chemical substances will usually come for consideration in one of three circumstances: the physician may be called to what is apparently a case of acute poisoning, which must be distinguished from an acute illness; he may be called upon to treat a case of illness which is in reality a case of slow poisoning; and in the course of certain medicinal treatments cumulative effects of known drugs may develop to toxic activity. Because of the distinctness of these three groups of cases, as the physician meets them, our consideration of poisons will be subdivided into three sections, corresponding as closely as possible to these groups. Even within each group, it is deemed advisable to subdivide the members of the group in such a way that simplification of diagnosis and treatment will be secured, even though it be necessary in doing so to disregard

the usual modes of classifying poisons. When one recalls the similarity of many different types of poisoning and their general resemblance to certain diseases, and remembers the importance of promptness of decision in diagnosis and treatment, the value to the physician of a grouping based upon the dominant, immediate symptoms will be evident.

The physician should bear in mind, however, that the classification which is most helpful to him may be of little value to the chemist whose aim is the detection of a poison after death of a patient, or to the pathologist whose post-mortem studies are carried out to determine the probable cause of a death which may have been due to some poison. For the use of workers interested in other than the clinical aspects of the subjects, various other classifications are in use. It is advisable that one should be familiar with the meanings of the terms used in the more important of these, and be able to place in its proper physiological class any of the more important poisons.

Reese follows the Physiological Classification:

(Definitions by present writer.)

I. Irritants—

True Irritants,—exert an injurious influence, which is followed by inflammation, upon the tissues they reach, whether before or after absorption.

Corrosive Irritants,—produce gross local destruction of tissue at point of contact.

II. Neurotics—

Cerebral:

Narcotics,—by depression of the cerebrum produce prolonged stupor after primary period of excitement.

Anesthetics,—by widespread action upon the central nervous system produce complete unconsciousness with only a short period of stupor before onset of coma.

Spinal (also called Tetanics),—by increasing activity of spinal cord tissues produce spasmodic or continuous muscular contractions.

Cerebro-spinal:

Deliriantes,—excite the activity of the cerebrum with production of disorder of the mental faculties.

Depressants,—diminish the physiological activity of various organs largely through action upon some controlling nervous structure.

Asthenics,—produce marked loss of muscular power, to extent of prostration, by depression of nerve centers, nerve endings, or intracellular metabolism.

Tanner follows a similar classification, but introduces two subdivisions of the True Irritants which merit note:

Simple Irritants,—those acting essentially before absorption.

Specific Irritants,—those acting after absorption, as before, and tending to show selective action upon certain tissues.

His use of the term “Asphyxiants,” referring to “noxious gases producing neurotic symptoms by means of blood poisoning,” is also convenient.

Perhaps the best illustration of a classification for the use of specialists in toxicology is that of Kobert, which is given here in condensed form:

- I. Poisons which Cause Coarse Anatomical Changes of the Organs.
 - A. Those which especially irritate the part to which directly applied.
 - B. Those which have a minor effect locally, but change anatomically other parts of the body.
- II. Blood Poisons.

Agglutinate or hemolyse erythrocytes, or oxidize stably or decompose hemoglobin.
- III. Poisons which Kill without the Production of Coarse Anatomical Change.
 1. Cerebro-spinal poisons.
 2. Heart poisons.
- IV. Poisonous Products of Tissue Change.

Still other classifications are made, according to the organs in which pathological change is typically produced, according to physical condition, according to chemical characteristics which are of value in the isolation or identification of poisons, and others upon the origin of the poison. Those given above are sufficient, in addition to that followed in the body of our discussion, for the physician who is not engaged in special work in the field of general toxicology. The physician should not forget, however, that every case of poisoning to which he may be called is potentially a medico-legal case. This will be evident if, noting the large proportion of poison cases which are of suicidal intent, one will recall the frequent legal actions in connection with the disposal of property or securing of life insurance of the supposed suicide and the importance of the attending physician's testimony in the case. Consequently, the physician is compelled to have a wider knowledge of the subject than would suffice for the medical management of the patient's condition. An outline of the

most important requirements of the physician in medico-legal matters is given in a later section.

It is of interest before entering upon the discussion of acute poisoning, where will be discussed most of the general principles of the subject, to know something of the frequency of poisoning. In all manner of ways toxicologists have sought to determine the actual frequency of these occurrences, but unsuccessfully. The nearest we can come to securing definite ideas of this question is by consulting mortality statistics to determine the number of deaths occurring from poisons. Even these figures are but an approximation of the truth. The following figures will convey some idea, however. They state the average number of deaths per year occurring in the Registration Area of the United States, comprising about two-thirds of the population of the nation, during the three years 1911, '12, and '13:

DEATHS PER YEAR

(In Registration Area of U. S., 1911-'13)

From Suicidal Poisoning.....	2845
Suicidal Asphyxia	1125
Accidental Food Poisoning.....	570
Accidental Acute Poisoning.....	1400
Chronic Lead Poisoning.....	150
Alcoholism (acute and chronic)....	3250
Other Chronic Poisonings.....	290
<hr/>	
9630	

From these figures it is evident that the frequency with which the physician is called upon to manage acute poison cases is relatively little; certainly much less on the average than to manage typhoid fever, for example. The infrequency of engagement in this branch of practice, combined with the necessity of prompt and correct action when the emergency of an acute poisoning presents itself, emphasizes the importance to the physician of having a certain minimum of information of toxicology definitely crystalized in form for immediate yet intelligent application. It is the aim of the following sections to present this. During the management of the case subsequent to moments of early urgency, the physician owes it to his profession, as well as to the patient, to consult in reference to the case either a thorough treatise upon the subject or, when possible, a competent toxicologist.

SECTION I.

ACUTE POISONING.

By Acute Poisoning we mean the prompt and marked disturbance of physiological functions caused by the action on the body of a single excessive dose of a drug, which may or may not result in death.

Chronic Poisoning is the gradually increasing, or persisting, disturbance of physiological function caused by the action of repeated small doses of a drug over a long period. Cumulative Poisoning is a special type of chronic poisoning characterized by the sudden appearance of symptoms resembling those due to the administration of a single excessive dose of the drug, but due in reality to the repeated administration of small doses.

Quoting from Witthaus (Toxicology, 1911): "The action of a poison or corrosive may result in death, in complete recovery, or in partial recovery.

"Death from the primary action of poisons or corrosives may be due to **exhaustion** caused by persistent vomiting, violent convulsions, or severe pain; to **cerebral paralysis** affecting the respiratory centers; to **cardiac paralysis**; to **edema** of the lungs or of the glottis; to **internal asphyxia** by modification of the hemoglobin of the blood; to **diminished body temperature**; to **hemorrhage** caused by blood vessels by corrosion; or to more remote effects, such as suppression of urine, etc.

"Complete recovery results in most cases of non-fatal poisoning in which the toxic agent is eliminated, and in which the pathological changes are either insignificant in kind, or of such a nature that progressive repair follows removal of the cause.

"Partial recovery is practically recovery from the primary effects of the deleterious substance, followed by prolonged illness or death from its secondary effects. Thus death from **starvation** frequently occurs months after the primary effects of the mineral acids and alkalies have disappeared; **atrophy and degeneration** of various organs follow as secondary effects of several poisons, of the gastric follicles by arsenic, of the liver and kidneys by phosphorus, of the extensor muscles by lead, etc.; **necrotic changes** are produced in certain organs, as in the maxillary bones by phosphorus, in the teeth by mercury, and in the extremities by ergot; **cataract** follows ergot poisoning, **amaurosis** is caused by wood alcohol; and many poisons cause as secondary effects great sensitiveness to external influences of certain organs, which persists for months or years."

The mode of action of drugs to produce these results, the processes of absorption and elimination of various drugs, and factors which affect the degree of action of drugs are rather studies for pharmacology than toxicology. However, for the intelligent management of any case of poisoning, it is necessary that these things be kept in mind. The more essential points will be briefly cited in connection with treatment. The following list of **factors which modify the degree of activity** of the poison will be of value in making the prognosis of any given case:

Size of dose.

Concentration of the drug in the preparation used.

Physical condition of drug (solid, finely divided, in solution).

Absorbability of drug.

Absorptive activity or reflex connections of tissues with which drug is brought into contact.

Age of individual.

Size of individual.

Temperament of individual.

Physical condition of individual.

Idiosyncrasy; or tolerance for particular drug (influenced by past habit).

Character and amount of stomach contents (when administration by mouth).

DIAGNOSIS OF ACUTE POISONING.

In acute poisoning the symptoms most frequently observed are abdominal pain, colic, nausea, vomiting, purging, collapse or shock, intense cerebral excitement, delirium, convulsions, stupor, coma. Of these there is not one which does not occur far more frequently as a result of other causes than from poisoning. Because of this fact, unless there is a definite history of the administration of a poison, the physician must first eliminate the possibility of the presence of an acute illness, before proceeding with such radical measures as are usually needed in case of poisoning.

Whenever a person apparently in good health suddenly becomes sick, experiencing sharp abdominal pain, vomiting or purging, or showing marked disorder of circulatory, respiratory, or nervous system, as indicated by any of the symptoms of the groups shortly to be outlined, the possibility of poisoning should be considered. In each case there should be rapidly called to mind the symptom-complexes of these diseases which are suggested by the patient's condition, and these compared with what is actually observed in the patient. As a very few minutes' delay may make the difference between life and

death of the victim if the case be one of poisoning, it is not possible to overestimate the importance of promptness of decision and of the immediate commencement of treatment as indicated, according to the particular poison which happens to be at work.

The following is suggested as an outline of the procedure to be followed in arriving at a working diagnosis as to whether the case is one of poisoning or of acute illness:

1st. Permit suspicion of acute poisoning to be aroused when a person believed to be in good health becomes suddenly sick.

2nd. Secure statement of those present with the patient of their knowledge or suspicion of poison having been taken.

3rd. Inquire as to the presence, in the house or elsewhere where patient might have secured it, of any poisonous substance suspected; for example, rat poison, ant paste, strong mineral acids or alkalies, oxalic acid for removing ink spots on cloth, any powerful drug commonly employed as a medicine.

4th. Inquire as to the exact time of onset of symptoms, and as to the recent ingestion by the patient of any food, drink or medicine. If possible, secure some of the substance for inspection for suspicious characteristics.

5th. If vomiting has taken place, secure the vomitus and inspect it for suspicious characteristics (odors of poisonous substances, blood, or blood-streaked mucus, altered condition of constituents due to action of some chemical substance, or even presence of the chemical itself). If at all suspicious in appearance, have it sent to a laboratory for examination at once, after giving what immediate treatment is indicated.

Inspection of mouth and pharynx may show signs of corrosion or marked irritation, and absence of the furred tongue so characteristic of gastro-enteric diseases.

Pain will persist after vomiting in most cases of poisoning. There may be pain in throat and mouth, which would not occur in cases of simple gastritis.

6th. Obtain history of previous occurrence in patient of convulsions; of possibility of epilepsy, diabetes, Bright's disease, cardiac disorders, advanced pregnancy, gastric cancer, headache, exposure to sun or excessive heat, recent falls or

injury. Take note of age and physical appearance of patient in reference to likelihood of presence of any of these conditions as a factor in producing his immediate condition.

7th. Note the order of occurrence, intensity, and rapidity of progression of the symptoms and compare them with those of simulating diseases. Determination of body temperature may be an important item in this connection.

8th. If no vomiting has occurred, or vomitus has been destroyed before inspection, and no decision is promptly reached, but conditions remain strongly suspicious, empty stomach and examine vomitus or washings.

If the case appears to be one of poisoning, the treatment must be governed by the nature of the poison. Tho the diagnosis of the particular poison from the symptoms alone is often impossible, it is possible usually to recognize the general type of the poison and to guide treatment along certain general lines that will meet the probable dangers of each general group. The following outline will be of value, not only in the recognition of the types of poisoning, but will be of service in suggesting the forms of sudden illness that may closely resemble or be closely resembled by poisons of the various groups. The list of poisons causing symptoms of each type is arranged as nearly as possible to indicate the relative frequency of death from the various causes, as indicated by mortality statistics in some of the Eastern States during the years 1877 to 1902. It should be borne in mind that the relative importance of the various poisons is continually changing and is different for each section of the world. At a time when more than 60% of the deaths from poisoning in France were due to either arsenic or phosphorus, these two poisons accounted for only 5% of the deaths in England; during the same twenty-six-year period lead compounds, opiates and carbolic acid in England accounted for 60% of the poison deaths, while in France these accounted for less than 5% of the deaths. There is an apparent tendency for a large proportion of the poisonings in any one locality to be due to certain few "popular" poisons. A knowledge of what those are in his community will be of distinct assistance to the physician in his poison diagnosis, just as knowledge of the existence of a certain epidemic assists him in his general diagnosis.

OUTLINE OF THE TYPES OF POISONING.

I. **Gastro-Intestinal Irritation.**

Usual Symptoms:

Sudden development of **gastric or abdominal pain**;
Nausea;

Vomiting, often persisting after original stomach contents have been evacuated and often then containing blood;

Purging;

Certain occasional special symptoms with certain drugs;

Shock or Collapse,—recognized by

Muscular weakness,

Profuse perspiration,

Clammy skin,

Rapid and feeble pulse,

Breathing labored and inefficient,

Fall of body temperature,

Anxious face if conscious,

Tendency toward loss of consciousness,

Coma.

Usually Caused by:

Mercuric Chloride,

Arsenic Compounds,

Carbolic Acid,

Lysol,

Phosphorus,

Lead Salts,

Oxalic Acid,

Potassium Chlorate,

Sulfuric, Hydrochloric, and Nitric Acids,

Ammonium Hydroxid, Caustic Alkalies,

Formaldehyde Solution, Antimony Compounds,

Zinc and other Metallic Salts,

Colchicum,

Croton Oil.

FOOD POISONING,

(Digitalis may strongly resemble this type, but is a very rare type of accidental poisoning.)

Simulated by:

Cholera Morbus,

Malignant Cholera,

Other Acute Gastro-Enteritis,

Peritonitis,

Ileus,

Strangulation of Hernia,

Rupture of perforation of Gastric or Duodenal Ulcer,

Rupture of Uterus or other Viscus,

Acute Pancreatitis,

Biliary Colic,

Renal Colic,

Onset of severe types of Smallpox, Scarlet Fever, Yellow Fever,

Hysterical Meteorism.

II. Central Nervous System Irritation.

Usual Symptoms:

Cerebral excitement, restlessness;
Increased reflexes, **muscular twitchings**, cramps;
Convulsions;
Delirium;
Skin, dry and hot, often flushed;
Pulse, rapid but full;
Breathing, rapid;
Body temperature often raised;
Pupils often dilated;
Often diarrhea;
In case of volatile oils, gastric pain, vomiting, gen-
ito-urinary irritation, abortion of pregnant
women, added to preceding;
Coma.

Usually Caused by:

Strychnin,	Cocain,
Volatile Oils,—Turpentine, Tansy, Pennyroyal,	
Wintergreen;	
Atropin,	Stramomium,
Caffein,	Hyoscin,
Camphor,	Cantharides,
Iodoform (sometimes),	
Salicylic Acid (sometimes).	

Simulated by:

Tetanus,	Meningitis,
Eclampsia,	Acute Mania,
Sudden onset of exanthematous fevers,	
Convulsions of Childhood, as in rachitic conditions,	
Hysteria,	

III. Vital Depression.

Usual Symptoms: less easily classified than in pre-
ceding types.

Physical weakness, due to

- (1) Irresistible drowsiness from cerebral de-
pression,
- (2) Peripheral nerve paralysis, or
- (3) Failing circulation (this form may resemble
collapse of Group I).

Often cardiac anxiety, if not stuporous;

Pulse, weak, rate slow, rapid or irregular;

Respiration, slow if in stupor;

Terminal coma, often developed early.

Many special symptoms occur in this group, which
are of greatest value in the recognition of the

specific poison. Several of this group, despite their very specific depression of a vital system, give rise to symptoms suggestive of gastrointestinal irritation, from which they must be differentiated by the accompanying specific action.

Usually Caused by:

Opium, Morphine, etc.	Digitalis,
Chloral,	Aconite,
Chloroform,	Ergot,
Ethyl Alcohol,	Veratrin,
Methyl Alcohol,	
Acetanilid and relatives,	Physostigmin,
Phenol, Cresol, etc., dilute	Pilocarpin,
Sulphonal, Veronal, etc.,	Muscarin,
Nicotin,	
Lobelia,	
Conium,	
Gelsemium,	
Hydrocyanic Acids and Cyanids,	
Nitro-benzol.	

Simulated by:

Apoplexy,	Uremic Coma,
Traumatic Shock,	Diabetic Coma,
Heat Stroke,	Hysteria.
Angina Pectoris,	
Epilepsy,	

IV. Respiratory Irritation or Asphyxiation.

Usual Symptoms:

Choking, violent coughing, or dyspnea;
 Congested face and some bulging of eyes;
 Certain special symptoms in each case;
 Usual termination in coma before death.

Usually Caused by:

Carbon Dioxid,
 Carbon Monoxid,
 Hydrogen Sulphid,
 Ammonia,
 Chlorine, *Wax gases*
 Bromine,

Simulated by:

Mechanical Suffocation,
 Foreign Body in Air Passages,
 Pulmonary Embolism.
 (Apoplexy, Epilepsy, and possibly rupture of an aneurism.)

DIAGNOSIS OF DEATH.

In connection with the mode of action of poisons we have already, in the quotation from Witthaus, cited the most important of the immediate causes of death. But a few more comments upon this subject are deemed advisable because of the importance of having a clear conception of the conditions surrounding this event. "In the interaction of tissues, the circulatory, the respiratory, and the nervous systems are indispensable, and the cessation of the activity of any one of them will bring about somatic death. Organs, too, at first sight insignificant, such as the adrenals or parathyroids, have been proven to be of prime importance. With so many organs essential to life, it is clear that there may be many ways in which somatic death may intervene because the exhaustion of one single system or organ may be enough to permit somatic death to occur." But in early death from poisoning the cause of somatic death will almost always be found to be in the failure of one of the first three mentioned systems, while the lingering deaths occasionally occurring after apparent recovery will be more frequently due to injury to one of the other essential, tho less prominent, functions of the body. The management of any particular case is so largely governed by the mode of death threatened, in severe cases of poisoning, that the phenomena associated with each mode will be discussed rather in connection with general treatment of cases of poisoning than in this paragraph.

There will be times when the physician, upon arrival, will find the victim either dead or apparently dead. In such events the physician should first assure himself, by the following tests, that the patient is really beyond possibility of resuscitation and then, if such be the case, give regard to the legal aspects of the situation which are dealt with in a later section of this outline. Except in cases where the patient is actually dead at the time of the arrival of the physician, no concern for the legal aspects should cause the physician to delay prompt action to combat the effect of the poison.

Herewith is appended an outline presenting the so-called

SIGNS OF DEATH.

(Adapted from Reese, Toxicology.)

1. Complete and Continuous Cessation of the Function of Circulation.

Determined by careful stethoscopic examination for heart sounds; by lack of change in color of tip of a finger which has been ligatured, which in event of continuing circulation will become pinker or purple.

2. Complete and Continuous Cessation of the Function of Respiration.

Determined by immobility of feather held before nostrils and mouth; by failure of collection of moisture upon mirror held before nostrils and mouth; by immobility of mercury in small vessel placed upon bared chest.

3. Change in the Appearance of the Eyes.

Failure of light reflex constriction of pupil.

Development of opacity of cornea.

Failure of "winking reflex" when cornea or conjunctiva touched.

Loss of elasticity of eyeball; depression or distortion caused by pressure remains instead of returning to normal contour, as in life.

Disappearance of rose-red color of optic papilla as seen on retinoscopy.

4. Pallor of the Body.

5. Loss of Animal Heat.

To be determined by thermometer. Twelve or more hours required for temperature of adult's corpse to fall to that of surrounding air.

6. Appearance of Rigor Mortis. May appear within an hour and may not occur for several hours. A positive sign when found.

7. Cadaveric Lividity.

Violet colored patches developing in the most dependent parts of the body, usually not before several hours after death has taken place.

8. Coagulation of the Blood in the Veins.

9. Spontaneous chemical disintegration of the Tissues.

Observations cited under the first three of the foregoing headings will usually suffice to answer the question as to death having taken place at the time of arrival at a case of poisoning. But the physician should bear in mind the possibility, in some of these cases, of maintaining artificially the ventilation of the lungs, when the process of respiration has temporarily ceased owing to depression of nerve centers which may recover from their depression if oxygenation of the blood be maintained; he should remember that in some cases of profound collapse where the heart has practically ceased to beat from lack of diastolic accumulation of blood in the auricles, prompt compression of limbs or abdomen may result in forcing forward along the veins and into the heart enough blood to sufficiently distend the auricles, that circulation may be resumed. In short, the physician must decide

whether it is likely that any of these three vital functions has been defunct long enough to have resulted in the death of the cells of the vital organs; and, if it is possible in any way to do so, he must resort to some mode of procedure which will reinstitute the conveyance of oxygen to the tissue cells, so that they will not die during a period when the body, unassisted, is unable to maintain this most essential of all functions.

GENERAL TREATMENT OF POISONING.

(Thruout this section dosage of drugs mentioned is the Adult Dose.)

In the management of any case of poisoning, there are prime objects toward which efforts must be directed. These are:

1. Remove the poison from the body.
2. Check the action of the poison, by the use of the proper antidote.
3. Assist the body in the maintenance of those functions which are indispensable to continued life, namely: oxygenation of the blood, conveyance of oxygen to the vital tissues by means of circulating blood, continued functioning of those portions of the nervous system which are essential to the continuance of respiratory and circulatory activities. There are certain symptoms which frequently develop as the result of the action of poisons, and which have a marked influence upon the foregoing three vital needs of the body. These will presently be discussed.

REMOVAL OF THE POISON.

Always in case of poisoning, the first indication in treatment is removal from the body of any unabsorbed poison. This is true whether the poison is in the stomach; in the intestines; in a wound, as in snake-bite; in contact with the tissues in the form of a medicinal dressing; in the lungs, as in asphyxia; in the rectum or vagina, as a suppository or an injection. It should be remembered that, except in the case of corrosive and simple irritant poisons, it is not the poison that is in the stomach or intestines that endangers life, but that which is absorbed into the system. It should be evident that if the poison can be removed from the point of absorption while still but a small quantity has gotten into the system, dangerous results will be largely forestalled. In fact, the body itself, in a great proportion of poison cases, promptly reacts with vomiting and purging, which tend toward the removal of poisons from the gastro-intestinal tract. Prevention of continued absorption of a drug, by chemically converting it into an insoluble or inert substance, is equivalent to removing

the drug from the body in many cases. Often this procedure is combined with those aiming at the physical removal of the poison, but the discussion of this mode of treatment belongs with that of antidotes.

Emptying the stomach is the most important single procedure in practical toxicology. It is accomplished either by exciting vomiting or with the stomach tube.

Vomiting is induced either by the use of an emetic or by tickling the throat and base of the tongue with a feather or the finger.

An emetic is a chemical substance whose internal administration is followed by vomiting. The most important with the mode of use are:

Powdered Mustard—a tablespoonful stirred into a glass of warm water.

Ipecac, Syrup of—one-half or one tablespoonful.

Common Salt—one tablespoonful in a pint of warm water.

Zinc Sulphate—15 to 30 grains completely dissolved in warm water.

Copper Sulphate—15 to 30 grains completely dissolved in warm water.

Warm Water—with addition of soap-suds, 2 pints or more, until result.

(Any of the preceding may be repeated if no vomiting follows in 15 minutes.)

Apomorphine Chloride—1/10 grain by hypodermic. Results in 10 minutes usually.

Emetics are inferior to the use of the stomach tube when this is available and can be used with safety. Two reasons justify this statement: first, the slowness and independability of their action on many cases; second, the fact that most emetics are themselves irritants and if their action is imperfect the residue remaining may add to the sufferings of the patient. To this may be added the statement that the vomiting set up may be exhausting and thus act to the patient's harm, while apomorphine is distinctly depressant to the vital centers of the nervous system. But there are times when the stomach tube cannot be used, as when a patient is unmanageable from delirium or convulsions, or when after a meal the stomach contains particles too large to pass thru the tube which is consequently blocked. So knowledge of emetics is essential. **That remedy should be used which can most quickly be secured.**

The stomach tube consists of piece of non-collapsible, but fairly flexible rubber tubing with a side opening near its closed distal extremity. On the tube should be marks indi-

cating the distance usually necessary to introduce it to have it reach the bottom of the stomach in an average-sized adult. By means of a short, straight piece of glass tubing, the stomach tube is connected with a more flexible piece of rubber tubing to the end of which is attached a funnel, preferably of flexible rubber. Each tube should be about three feet in length, the stomach tube usually being introduced to a distance of 20 to 24 inches, the distance being measured from the point where the patient's incisor teeth touch the tube. In some cases there is a suction bulb fitted between the stomach tube proper and the funnel tube; but it is questionable if it adds any advantage in emptying the stomach when the use of an abundance of water is permissible. The stomach pump, formerly more widely in use, consisted of a rather stiffer tube than the one now designated a stomach tube and had attached to its upper extremity a large piston-syringe and a pair of valves, by the manipulation of which the contents could be forcibly extracted.

To empty the stomach with a stomach tube, the patient is placed in a sitting position and the head is slightly bent forward, not back. A gag of some sort to prevent the patient's biting down on the tube should be at hand. After thoroly wetting the tube with warm water,* it is grasped by the fingers of the right hand about seven inches from the stomach end, being held in about the manner that a pencil is usually held; about six inches back of the right hand, the tube is more firmly held by the fingers of the left hand, a most convenient position being to place the tips of all the fingers on the convexity of the tube and the thumb opposing them on the concave side of the tube's curve. Standing on the patient's right side, when the tube is held as above suggested, the patient is directed to open his mouth by dropping his jaw, not by throwing his head back. (In case the patient is unconscious or resisting, the jaw will have to be forced open by an assistant.) The tube is then passed straight back into the pharynx, care being taken to go to one side of the uvula if possible, and while it is held steadily against the posterior wall of the pharynx, the patient is directed to swallow. (By this it is meant, not to press forcibly, but to avoid rubbing the end of the tube around over the pharyngeal wall, which would tend to excite gagging.) The tube should be held lightly enough by the fingers of the right hand so that, when the pharyngeal muscles grasp it and com-

*Altho in general practice, when the mucous membranes are normal, warm water seems better for lubricating the tube, when the membranes are corroded or inflamed, warm oil is preferable. In addition to first drying and then oiling the tube, $\frac{1}{2}$ to 1 ounce of warm oil should be introduced thru a catheter into the upper part of the esophagus or the post-laryngeal region of the pharynx.

mence to carry it into the esophagus, the movement of the tube will not be checked, the left hand following up the movement of the tube into the esophagus and preventing the pull of the free end from drawing it out. As soon as the tube is grasped by the esophagus, it is to be slowly but firmly forced down, without any further effort to swallow being made by the patient, until it is well into the stomach as indicated by the marks on the tube. The tube now being held in place to prevent either its expulsion or its being swallowed, the funnel tube is connected to it. Thru the funnel, about six or eight ounces of warm water (about 110° F.) are introduced to the stomach. While the tube is still filled with water, its upper end being pinched to retain it, the funnel is lowered into a basin which is held or placed about a foot below the level of the patient's stomach. The contents of the stomach should be siphoned out into the basin. This process of introducing fluid into the stomach and siphoning it out should be repeated until the washings come away clear. As will be cited later, this method is often utilized for the application of a chemical antidote to the walls of the stomach. **The process of washing out a stomach in this manner is designated "lavage."**

In case of slowly absorbed poisons, they are often carried into the intestines where absorption will continue as long as they remain. **To empty them from the intestine**, the body often displays rather prompt purging. In case this does not take place spontaneously, it is usually advisable to **administer a cathartic drug, or to flush out the colon**, or do both. Of the drugs for this purpose those chosen should be rather rapid in action. Probably the three following come the nearest to meeting the requirements:

Castor Oil—one or two tablespoonfuls. Seems to be contraindicated in case of Phosphorus, Cantharides, Carbolic Acid, because of increasing absorption.

Sodium Sulphate—one rounded teaspoonful (2 dr.) dissolved in water.

Magnesium Sulphate—two rounded teaspoonfuls dissolved in water.

When the poison is in a wound, incision to aid free bleeding of the area, compression of the part between the wound and the heart, flooding of the wound with slightly hypertonic salt solution may be resorted to.

When a medicinal dressing is the cause of poisoning, of course, the dressing should be removed, and moist dressings applied in such a manner as to "draw" from the tissues the fluids which may contain some of the drug.

When the lungs contain the poison, removal of the patient to fresh air and performance of artificial respiration constitute the procedure.

When the poison has been administered per rectum or per vagina, these structures should be cleansed by copious washings containing, if possible, a chemical antidote.

Altho they might be considered under the heading of physiological antidotes, there may be mentioned here those measures which may be used **to remove the poison from the system after absorption.** Formerly it was quite common to administer for this purpose the stronger diuretic drugs. The later tendency is to spare the kidneys any unnecessary irritation. Drinking abundantly of hot water, together with the application of mild diaphoretic measures, such as administration of a hot foot or leg bath, while the rest of the body is kept warm, will usually excite copious perspiration which would assist in the elimination of alcohol, lead, mercury and some other poisons. More vigorous sweating measures may be used,—electric light bath, hot blanket pack, etc.,—but discretion should be used not to aggravate a patient's exhaustion, and any such treatment should be accompanied by the drinking of an abundance of water and should be followed by some momentary tonic cold application.

A most radical procedure for the removal of absorbed poisons has been resorted to in some instances: **bleeding, to remove some of the poison contained in the blood,** followed by intravenous infusion of a physiologic solution (saline: preferably Ringer's) in quantity double the quantity of blood removed. This procedure might act favorably by accelerating the addition of new erythrocytes to the blood stream from the hemopoietic organs, as well as by increasing elimination thru the usual channels because of increased blood volume and dilution. It is reported to have been found of benefit in poisoning by hydrocyanic acid, carbon monoxid, potassium chlorate, and other true blood poisons, in experiments upon animals.

ANTIDOTAL TREATMENT.

Checking the action of a poison, accurately speaking, must be done before the drug gets into the tissues. In this statement, it should be distinctly understood, we refer to the checking of what in pharmacology is designated the primary action of a drug, not merely offsetting the effects of the action of the poison. As an illustration of what is meant, the administration of atropin after a drug which stimulated the vagus center would stop the slowing of the heart due to the first drug, by depression of the vagus endings in the heart; but the stimulation of the vagus center would nevertheless be continuing.

Most frequently we mean by the expression, checking the action of a poison, the use of some agent which will interfere with or prevent its absorption, or by partial oxidation or other chemical change destroy its physiological activity.

An Antidote is a substance which will check the action of some specific poison.

A Chemical Antidote is a substance which checks the action of a poison by acting directly upon the poison, and so altering it that it becomes insoluble, and therefore impossible of absorption, or otherwise renders it inert.

A Mechanical Antidote is a substance which acting physically interferes with the absorption of a poison or otherwise checks its action.

A Physiological Antidote is a substance which acts upon the tissues of the body in a manner which prevents the injurious effects of poisons; or, in a less exact sense, a substance which excites functional activities which offset the effect of a poison.

Chemical antidotes usually act upon a poison before it has been absorbed into the blood stream. There is a widespread, but erroneous, belief that for every poison there is a definite counter-poison. While it is true that there are methods of altering to a harmless form practically any poisonous substance when it is being dealt with in a test-tube, there are practically none which can be so altered after their absorption into the system, excepting in so far as the body is able by its normal activities to bring about these changes. The body itself does however possess the ability to chemically alter and render inert many poisons, as illustrated by the oxidation of many of the alkaloids and of phosphorus and the active sulphids, the fixation in the tissues of the liver of some of the alkaloids and many of the heavy metals thus delaying their action upon other more sensitive tissues, the neutralization of acid intoxicants by the basic products of protein metabolism. A good illustration of the application of the older belief that chemical antidotes could act in the system after absorption of the poison and of antidote is found in the former custom, now abandoned, of administering alkalies in cases of poisoning by chloral hydrate, with the hope that the alkali would cause the disintegration of the poison to chloroform and a formate.

The mechanical antidotes of importance are animal charcoal, albumen, starch paste; gum arabic solutions. They act essentially by themselves absorbing from solution the poisonous substance, thereby interfering with its absorption into the blood stream. They also interfere with the free diffusion of the dissolved poison throughout the stomach or intestinal

contents and thereby delay absorption. But they do not cause any destruction or alteration of the poison, and do not permanently prevent its absorption. Therefore they are not permitted to remain in the gastro-intestinal tract, but **are removed usually by lavage after allowing a short period of time for their action.**

The physiological antidotes of real value are few. This is because no two drugs possess completely antagonistic properties. Many which might help to quell some of the disagreeable symptoms of a certain poison would be found to augment other symptoms. "Physiological antidotes are of value only when they tend to counteract an effect of a poison which is dangerous to life." There is a marked tendency for toxicologists to avoid the use of physiological antidotes as much as possible and to substitute for them physical procedures for the management of symptoms. Illustrating this tendency we find in Witthaus the statement, "There are many cases reported in medical literature as cases of morphin poisoning which are in truth poisonings by atropin and morphin, the former alkaloid having been administered as a physiological antidote."

Because of the fact that antidotes must be used in the most specific manner, if they are to be of value, their proper employment involves recognition of the exact poison or chemical group to which the poison belongs. A summary of specific uses of the important antidotes will therefore be more appreciated after the discussion of the various individual poisons, where such a summary will be found.

As a **general antidote**, for use when the specific poison cannot be promptly determined, **Magnesia, one tablespoonful; powdered Charcoal, two tablespoonfuls; mix to a thin paste, stir into a pint of warm water; give one-half of a tumbler full every ten minutes until all taken, then empty stomach at once.** The stomach should, if possible, have been emptied before the use of the antidote, so the action can be exerted upon the poison that is in contact with the stomach wall.

SYMPTOMATIC TREATMENT.

The **symptoms** which present themselves during the course of a case of poisoning are almost without number. Many of them need be given no attention after noting their significance. The following conditions, **when they threaten the safety of the patient, should be promptly relieved by thera-**

peutic procedures applied with a vigor determined by the urgency of the symptom:

Collapse or Shock.

Pain, and

Fright, or excessive anxiety, which contribute to the preceding.

Syncope, or more particularly the underlying Circulatory Failure.

Respiratory Failure,—

External, due to

Central Nervous System depression,

Paralysis of voluntary motor nerve endings,

Spasmodic contraction or fixation of respiratory muscles,

Exhaustion,

Edema or spasm of the glottis, or

Pulmonary Embolism.

Internal, due to

Circulatory Failure,

Hemolysis or alteration of Hemoglobin.

Convulsions.

Delirium.

Coma.

Suppression of urine, due to circulatory changes or renal irritation.

Uremia.

Acidosis, due to

Hepatic degeneration, or

Absorption of acid poison.

Gastro-enteritis, or peritonitis.

Persistent Vomiting.

Persistent Purging.

Exhaustion, due to operation of any of the preceding conditions.

In the management of the foregoing, there may be needed knowledge of the technique of the following procedures, which can be only listed here:

Intravenous Infusion.

Hypodermoclysis.

Proctoclysis.

Blood Transfusion.

Tracheotomy.

Intubation.

Gavage. Lavage has already been described, page 21.

Rectal Feeding.

Hydrotherapeutic and other physical measures, (demand correct technique or results may be even the opposite of the ones desired).

Hot leg pack,	Hot fomentations,
Hot blanket pack,	Moist abdominal bandage,
Cold mitten friction,	Hot trunk pack.
Neutral bath,	
Electric light bath,	
Enemata,	

Osteopathic manipulations.

Compression of limbs and abdomen by bandaging.

Artificial Respiration.

Shaefer Method, not only least fatiguing but most effective.

Sylvester Method, of value when other reasons make supine position of patient preferable to proneness.

Faradic Method. (Neutral electrode applied over tip of xiphoid process and epigastrium; stimulating electrode (double) over phrenic nerves, close to posterior margin of sterno-cleido-mastoidei about one and one-half inches above the clavicles, circuit closed for two seconds, then opened for two seconds. Compression of lower thorax during open period.)

By Pulmotor or Lungmotor.

Intratracheal Insufflation.

Other measures will be discussed in connection with their uses.

Shock and Collapse will be thoroly discussed after outlining the procedure with the less complicated symptoms.

After removing and antidoting the poison in any case, the next most important procedure, in case no other urgent symptom is presenting itself, is the **control of the conscious suffering** of the patient, whether it be due to fright, excessive anxiety, or physical pain. This step is of greater importance than might seem the case, since all of these conditions increase tendency toward shock and collapse. The manner of the physician and his dominating the whole field of activity and everyone present with a spirit of certainty of the patient's ultimate welfare, are important factors in the management of the situation, not only when the patient is conscious because of its sustaining influence upon him, but because in such an atmosphere only can the physician secure the best available assistance from those present.

Pain control should first be sought by local measures, by **Chemical Antidotes**, and

Demulcents, or agents which mechanically protect and therefore soothe mucous membranes to which they are applied, are often effective. For this purpose may be used:

Thin starch paste, drank freely;

Egg albumen, the whites of half a dozen eggs beaten for a few seconds;

Mucilage of acacia;

Olive, cottonseed, linseed oil (good in alkali poisoning, bad in phosphorus and phenol).

Honey, flaxseed-tea, gelatin solution, and other bland liquids.

Chipped ice swallowed in small pieces often eases pain in throat and stomach.

When local measures fail the following can be used:

Morphin sulphate, $\frac{1}{4}$ grain, hypodermically, unless distinctly contra-indicated. (One-eighth grain may be used for relief of anxiety when no pain.) Or,

Hydrotherapeutic measures properly utilized are very sedative to abdominal pain. Of most value, hot abdominal fomentations, hot trunk pack, moist abdominal bandage. Care is needed to avoid burning patient.

Osteopathic manipulations applied to reflexly contracted spinal muscles, while patient lies as easily as possible on his back, are often a decided relief in abdominal pain in other forms of gastro-enteritis, and the writer has seen it beneficially used in a toxic case. The general principles governing the manipulative relief of distress and pain in other conditions may be applied in cases of poisoning after having given attention to the specific needs of the case.

Syncope, or fainting, a transient loss of consciousness from cerebral anemia following reflex production of circulatory incompetency. Usually has an emotional origin and is usually not of serious import. "A brief period of giddiness is followed by complete unconsciousness, the individual falling in a lax heap, often without time to hold on to any support or to make any preparation. The face is blanched, the pulse small, rapid and at times almost imperceptible." The recovery is usually spontaneous; it is assisted by slightly lowering the head and raising the limbs, administering ammonia gas from ammonia water, or smelling salts, by inhalation; friction of arms and chest, using a cloth wrung out in cold water and following with a coarse towel. These methods act partially by assisting the circulatory system to resume its normal tone and partially arousing the patient from the reflex cerebral inhibition that is a factor in the condition.

Circulatory Failure is the most serious of the ordinary symptoms found in connection with poisoning. In addition to the special types of circulatory failure associated with **Shock and Collapse which are later** (p. 34) discussed, there are cases in which the circulatory weakness is essentially dependent upon a poisoning or degeneration of the heart. In these cases a strain of any sort may arouse symptoms closely analagous to those arising from acute failure of the heart to properly discharge blood into the arteries, as in failing compensation. In describing specific poisons we shall use the expression **CARDIAC FAILURE** to apply to direct toxic or exhaustion injury to the heart muscle or its intrinsic regulating and conducting systems. **The symptoms** of this condition include varying degrees of the following:

Irregular heart beat, both as to force and to rythm; delirium cordis;

Pulse irregular in rate, strength, and volume, but tending toward lowering of tension and acceleration of rate;

Precordial distress or pain, even becoming anginal;

Dyspnea,—all accessory respiratory muscles in use in sudden failure;

Cyanosis, frequently;

Anxiety or Fear, causing first vaso-constriction and muscular tension, later

Muscular Relaxation and Cold Perspiration, as in Collapse.

Treatment of the condition should be designed to

Establish self-control in the patient in the crises;

Relieve the heart of as much strain as possible; by

Relaxation of the peripheral circulation, with skin well warmed;

Providing absolute rest of the patient;

Reduce excessive activities of heart which tend to further exhaust it.

The Procedures which will secure these results:

Reassure the patient by word and manner; **morphine**, $\frac{1}{4}$ to $\frac{1}{8}$ grain, **may be needed**, and is rational if patient's suffering cannot be stopped in other manners, tho it is less frequently so when the following procedures are well applied.

Place the patient in as comfortable position as possible, the horizontal position preferable if patient can secure respiratory comfort in this position. Do not allow patient to exert himself.

Hydrotherapeutic measures to control the excessive activity of the heart and secure peripheral relaxation of the blood-vessels,—(of value before collapse condition reached).

Wrap the patient in a well-warmed blanket and apply to the precordia a small wrapped ice-bag, to check irritability of heart. Every four minutes or so, remove the ice-bag and warm the skin by a brisk friction.

Give to each arm, leg, and thigh, in turn, a hot fomentation, followed by a cold mitten friction; then dry with rough towel and follow with bare hand friction until part red and warm. Do not allow patient to make any exertions during the procedures, and keep him well covered with warm blankets, except for part being treated.

If the condition is so severe that the patient is forced to retain an upright or leaning-over position for respiratory comfort, before giving leg and arms treatment, administer hot fomentation, cold mitten friction and bare hand rub to the whole length of the spine.

(These procedures are recommended by Abbott as being far superior to digitalis, strychnin and nitroglycerin, even in those severe cases where there has been sudden appearance of marked moist rales from acute pulmonary edema.)

During a less urgent but more prolonged condition of cardiac weakness, a wrapped ice-bag should be kept on the precordia during about 15 minutes out of every three hours, the patient meanwhile being kept always warm, and two or three times a day a mildly applied treatment similar to the preceding administered, omitting the fomentations and cold mitten to the spine.

Osteopathic manipulation for the relaxation of the contracted muscles in the upper dorsal and cervical regions, given gently, firmly, and slowly, are a distinct assistance to the patient's improvement, in all cardiac defects.

(If the physician is unable to give the foregoing attention to the patient he may substitute for the hydrotherapeutic and osteopathic measures, the routine drugs—nitroglycerine, 1/200 grain, repeated twice at 15-minute intervals, or until throbbing frontal headache shows physiological limit to be reached; or **when patient nearly prostrated**, too weak to assist respiration with axillary muscle group as in less severe cases, with none of the periodic increase in pulse tension seen in the struggling condition, hypodermic of 10 cc. **camphor in sterile oil**, or intramuscular injection of an ampoule of sterile **ergot**, or atropin 1/100 grain, and **later** for subsequent effect, if not contra-indicated, German **digitalin** 1/6 grain every four to twelve hours.)

See Collapse Treatment on page 38 for adequate management of collapse stage of cardiac failure.

During the management of the case subsequent to the early moments of urgency, the physician owes it to his patient

to protect him particularly against injury of a taxed circulatory system. Anticipation of acidosis and uremia are a part of this protection, and the care to have the diet a properly nutrient and digestible one. Taxing the heart with unnecessary drugs is not pardonable. But of all measures REST is the prime one. All exertion and excitement must be forestalled; comfort must be supplied to the patient in every possible way; and every effort exerted to give the patient a full period of normal sleep daily. Here the hydrotherapeutic and manipulative procedures will without question be found the most satisfactory. **A prolonged period for recuperation must be insisted on if it is known that the patient's heart was exposed to any primary poisonous actions.** During the convalescent period there should be daily manipulative, hydrotherapeutic and passive movement treatments given to gradually develop the heart's strength without allowing any opportunity for the patient to overtax it.

RESPIRATORY FAILURE is of so many different types that each must be dealt with differently according to the specific needs. A warning needs to be given, that prolonged application of artificial respiration measures, if too vigorously given, may result in traumatic pneumonia.

When due to central depressant,—

Oxygen Inhalation (with 3% to 5% carbon dioxide) to permit oxygenation of blood with the poor pulmonary ventilation; or

Artificial Respiration;

Cold Towel Rub to upper thorax and supraclavicular region.

Osteopathic manipulations to increase spinal cord tone thruout;

Atropin, 1/100 grain, repeated; or caffein and sodium benzoate 2 grains hypodermically; or coffee, by mouth or by enema, ½ to 1 pint, or more;—all recommended by most authors, but certainly not to be resorted to while the patient's condition shows signs of improvement by other methods, because of increasing incoordinate actions of the nervous system and adding to toxemia.

When due to Paralysis of voluntary motor nerve endings,

Artificial Respiration by mechanical means or intratracheal insufflation method are the only procedures effective, and these must be carried on until effect of the drugs has been recovered from, even tho it requires many hours.

When due to Spasmodic Contraction or fixation of respiratory muscles,—

Intratracheal Insufflation if possible without setting up excessive reflex convulsions,

An antidotal drug to check spasms by depression of motor centers of cord, as chloroform by inhalation or chloral per rectum (30 gr.) in strychnin poisoning; or hydrotherapeutic procedures, neutral bath to control convulsions.

When due to Exhaustion,—

Artificial Respiration by mechanical methods, selecting the method most agreeable to patient, if he is conscious.

When due to Edema or Spasm of the Glottis,—

Intubation, or

Tracheotomy, or

Intratracheal Insufflation.

When due to Pulmonary Embolism,—no known successful measures if embolus involves large area of distribution of pulmonary artery. May try

Oxygen inhalations, or

Intratracheal Insufflation.

When due to Circulatory Failure, treat this condition as indicated.

When due to Destruction of Oxygen-carrying Capacity of Blood,—

Transfusion of Blood, only available measure—this uncertain.

CONVULSIONS are caused in poisoning in several different manners. They may be asphyxial, due to failure of external or internal respiration; they may be due to irritants or stimulants acting directly upon the tissues of the cord or motor centers of the cerebrum; or they may be reflexes to excessive gastro-intestinal irritation; or be secondary to uremia or acidosis. They must be controlled when possible, for they tend to result in exhaustion if they are severe. **Management** depends upon recognition of the cause. The remedy may be found therefore among the following:

Artificial Respiration or Oxygen Inhalation.

Specific depressant drugs or physiological antidotes.

Specific treatments for Uremia or Acidosis. Or the following

Sedative Hydrotherapeutic measures,—

A full neutral bath, beginning 98° F., increasing to 102° F., while

Ice-bag (or cold affusions) is applied to head. Bath room must be warm and tub covered with sheet.

Manipulative treatment to upper cervical or sub-occipital region often of marked value, where convulsions not due to a powerful tetanic.

DELIRIUM is caused by a set of influences similar to those producing convulsions, but acting more specifically upon the cerebral cortex. Management of the condition is needed to give protection against exhaustion and is directed along the same lines as the management of convulsions. Drug methods of checking delirium are to be used with caution because of the danger of aggravating the injury to the cortical neurones. The drugs usually used are the coal tar antipyretics, chloral the bromids, or hyoscin (in acute mania). Better, and usually satisfactory, if properly applied, are the **Neutral Wet Sheet Pack**, or the **Neutral Bath**, as used in convulsions. **Osteopathic manipulations** to restore better circulatory conditions to the head often control the condition satisfactorily. **An ice-bag** to the head is advantageous if there is any elevation of temperature or of blood-pressure.

COMA, the terminal stage of most cases, is due to cessation of cerebral activity and loss of sensitiveness, making it impossible to arouse the patient. **Its cause** must be recognized and treated, whether it be **circulatory failure, exhaustion, acidosis, uremia, or toxic depression** by direct action of the drug. It is difficult to see what is to be gained by a procedure which compels consciousness by surmounting the insensitiveness of the brain or by chemical stimulation of the cells. The aim in the management of this condition should be simply to give necessary assistance to the circulation, respiration, excretion and metabolism, that life in the cerebral tissues be maintained until the injurious influence has been eliminated and the cells have recovered from their injury. No new injuries in the form of exhausting stimuli or protoplasmic poisons to the cerebral cells seem desirable. **Maintenance of body warmth** is one measure particularly important in this condition. This accomplished by means of hot water bottles, ordinary bottles containing hot water, hot sand bags, or hot salt bags, hot bricks, hot stones, hot irons, etc., all well wrapped to prevent burning the unconscious patient, and used in large number of moderate temperature rather than a few at high temperature.

SUPPRESSION OF URINE should be anticipated in those cases of poisoning where it is likely to occur, and should be treated if possible before **UREMIA** develops. **Guard against** in poisoning by Mercury, Phosphorus, Oxalic Acid, Cantharides, Turpentine. The causes of the suppression may be either action of the poison upon the renal epithelium or en-

dothelium, or development of passive congestion of the kidney. **Hydrotherapeutic measures supply the best method of managing the condition.**

Hot Abdominal Pack, or

Full Hot Blanket Pack, terminated in 20 or 30 minutes by tepid sponging, or cold mitten friction, with drying and bare hand friction, before wrapping in warm blankets to continue sweating for an hour or so. Mode of termination determined by circulatory condition of patient. While in pack patient should drink freely of water, and should have an ice-bag over the precordia and lower third of sternum. The head should be kept cool with an ice-bag. Hypodermoclysis may be used if patient unable to drink.

Full Hot Bath, may be used instead; or in less severe conditions, or where facilities poor, a Hot Foot Bath and Hot Fomentations to spine.

Intravenous infusion of saline solution by reducing toxemia and diluting blood sometimes establishes diuresis.

Any other rational diaphoretic measure may be used.

ACIDOSIS, when it occurs, is probably dependent upon toxic disturbance of liver or other organ influencing metabolism, or to direct absorption of an acid poison. It must be **controlled** if it makes itself at all evident. The **symptoms** by which the condition is recognized are not definite, tho they include **dizziness, dyspnea, restlessness, vomiting not due to gastro-intestinal irritation, muscular twitchings, tendency toward coma.** The best test for acidosis may be difficult to carry out in cases of poisoning: if the urine remains acid after the administration for two days of three doses of two drams each of sodium bicarbonate in water, taken on an empty stomach, there is likely some acidosis. This test might be of value during the subsequent care of many poison patients to avoid the insidious development of the condition. Many times it can be readily recognized. The **treatment** may be **administration by mouth of sodium bicarbonate** in large doses; **proctoclysis or intravenous infusion** of compound saline solution (see Collapse Treatment) to which is added one-half to one per cent of sodium bicarbonate; or (more recently recommended) 10% sterile solution of dextrose by proctoclysis or slow intravenous infusion. **Diaphoretic measures** may be an adjunct to this "blood lavage," as it has been called.

Persistent Vomiting and Persistent Purging need to be controlled if possible because they **tend toward production of both collapse and exhaustion.** Their control is not easy, however. Often they are due to collapse or to acidosis or uremia which must be treated. If primary, **lavage, flushing of the colon and the use of demulcents** may succeed. If there are no

contraindications, bismuth subcarbonate or cerium oxalate as protectives may be used in 30-grain doses. Cocaine hydrochlorid, $\frac{1}{4}$ -grain, as a local sedative, or a central sedative such as chloral, codeine, or the bromids are frequently recommended, but should be used with especial discretion in poison cases where they might have a synergist action upon some existing condition. **The local sedative hot abdominal pack, moist abdominal bandage, etc.,** may give the best results, particularly if supplemented by some general sedative hydrotherapeutic or manipulative procedure. Swallowing of **chipped ice** sometimes checks vomiting.

Exhaustion, of course, is to be managed by rest.

Care should be particularly exercised to have the patient properly nourished during his convalescence, the food being of a sort that it is readily absorbed, yet does not lack the proper constituents. After severe gastric irritation, it may be necessary to resort to rectal feeding until the stomach is able to resume its function. **Gastro-enteritis**, as it occurs after corrosive poisoning, presents a condition whose recovery demands the most intelligent care. The continued use of oils or other demulcents to **prevent adhesions and strictures**, in so far as possible, will be needed. Later, lavage to keep the stomach clean and free from accumulated exudates. The possibility of the development of peritonitis and the necessity of its management must also be kept in mind.

SHOCK AND COLLAPSE.

It seems, perhaps, out of place to introduce a discussion of shock and collapse in a manual of toxicology. But it is deemed wise to do so because these conditions are of such frequent occurrences in cases of poisoning.

For the rational management of these conditions, it is essential to have an understanding of the underlying alterations of physiological functions. At present it is not possible to secure from the various investigators of the subject even definite agreements as to the differentiation of these two conditions. Particularly, is it difficult to find any adequate discussion of the nature of these conditions of prostration as they are observed in cases of poisoning. Collapse is the term usually used to designate it in toxicology, whereas there are really present the combined conditions variously named shock, collapse and exhaustion. It makes little difference what the condition is called if it can be recognized together with the factors in its causation and the physiological perversions underlying its various manifestations. Whatever the order of development of the condition its recognition is by the same signs, once the condition is present.

The symptoms are:

Profound muscular relaxation;

Loss of volition;

Sensibility reduced;

Apathetic, much delayed, but rational answer to questions when aroused;

(The foregoing constitute the symptoms of impending shock, and may develop before circulatory weakness. Shock may be aborted by treatment.)

Face blanched, eyes and cheeks sunken, lips often blue;

Skin cold, usually bathed in perspiration;

Pupils dilated;

Breathing irregular, inefficient, labored, or Cheyne-Stokes type;

Breath cool;

Body temperature lowered;

PULSE RAPID, WEAK, SMALL.

Sometimes vomiting or retching.

Tendency toward coma or recurring syncope.

(In the condition designated collapse in this manual, the early mental lethargy of shock may be absent; and the pulse, due to relaxation of arterial wall, tends more to be soft than simply small.

With this group of symptoms we recognize two essential physiological perversions,—arrest of function of the central nervous system, and circulatory inefficiency. Adami chooses to designate the two conditions according to the priority of one or the other of these perversions, calling it shock when the central depression is initiatory; collapse when circulatory weakness precedes and induces the nervous depression. This would imply that shock is usually of rapid onset, collapse of slow onset, a differentiation insisted upon by some authors. Mummery, on the other hand, insists that collapse includes that group of prostrations due to reflex depression of the circulation, conditions which, like syncope, are of very rapid development. So much confusion exists regarding the naming of these conditions (altho the underlying perversion is different and requires consideration in treatment), that the writer in this discussion will endeavor to uniformly use the terms as specifically as possible to refer to definite types of prostration.

“Shock is a condition of ‘badness,’ induced by injury, and producing its effects by nociceptive (=potentially painful) afferent impulses to the central nervous system.” (A. Rendle Short.) Pain, then, is the essential antecedent of that form of

prostration which we shall designate shock; the term "pain," however, will be understood to include those agonizing "psychic sensations," such as extreme fright, fear, apprehension. "Shock" will imply, in consideration of the argument below presented, an underlying loss of spinal cord tone due to some perversion of the controlling impulses from the region of the medulla or midbrain, contributing to a circulatory inefficiency which may have a medullary origin, and accompanied by reflex cerebral depression. Whatever nerve center exhaustion may be present is considered to be limited to the medullary region and to be not primarily that of the vasoconstrictor center, tho this may be involved. Stimulation to affect the spinal centers, and of a non-painful nature, is therefore not contraindicated in this condition and may act to free the cerebrum from its dominance by lower centers excited by the pain, in addition to rousing the latent activities of the cord centers. Strychnine is absolutely contraindicated, because it opens up abnormal pathways for afferent impulses and results in purposeless, incoordinate, and excessive motor responses.

"Collapse" will imply a less definite type of prostration due to primary circulatory inefficiency which may originate in oligemia from hemorrhage or vomiting or purging, in exhaustion or toxic paralysis of the circulatory mechanism in any one of its parts, or in general muscular relaxation from the exhaustion of the muscular system itself, or those spinal centers which are necessary to its tone, the circulatory inefficiency secondarily resulting in cerebral depression tending toward coma. It will later be indicated that **shock may spontaneously progress to collapse.**

The management of collapse will, therefore, involve decision as to the particular need of the body in the case under consideration, with particular care to refrain from imposing new tasks upon the body in cases of exhaustion. Those rather pronounced cases of prostration following a blow upon the abdomen or the testicle are rather transient forms of shock, transient because the stimulus is active for but a short time, but occasionally inducing so complete a reflex checking of the circulation that it is possible for death to result.

MANAGEMENT.

To prevent shock, or to reduce severity, stop painful impulses. Morphine merely prevents their reaching consciousness, and checks only "psychic pain."

The following treatment for early shock is distinctly contraindicated where the prostration is due to exhaustion from vomiting, purging, convulsions, prolonged over-action of circulatory mechanisms, or to systemic primary toxic action upon the circulatory organs.

TREATMENT OF TRUE SHOCK (due to pain of corrosion or strong irritant action, or to fright, and accompanied by loss of spinal tone and spinal reflexes), **BEFORE COMPLICATED BY COLLAPSE.**

After emptying stomach, if indicated, and administering antidote

With patient's body horizontal, hips and legs raised to facilitate gravitation of blood to thorax but avoiding hypostatic venous congestion of head, direct the immediate and simultaneous application of the two hydrotherapeutic procedures.

Give hypodermic of morphine only in case patient is consciously suffering.

Give intravenous infusion of saline solution if pulse does not improve during hydrotherapy. (Hypodermoclysis or proclysis of little value if patient is nearly pulseless from other cause than fall of blood volume.) Adrenalin not indicated.

Keep patient warm, and inspire confidence.

Hydrotherapeutic Measures.

(At least three persons required to apply.)

I.

Very hot fomentation to Thorax, 30 seconds.

Immediately give ice rub, wiping off water formed as rapidly as it appears, and promptly rub dry with a coarse towel.

Repeat three or four times beginning with hot fomentation.

Then apply well wrapped ice-bag to precordia and keep chest warm.

Be very careful not to burn patient with fomentations.

Be very careful to keep parts warm after treatment, but do not weight down patient's chest with covers.

If necessary, repeat the above measure in 30 or 40 minutes.

Usually less vigorous methods will suffice for the later applications, as vigorous rubbing with bare hand or, if skin remains well warmed and pink, a cold (not ice) mitten friction.

II.

Apply hot fomentations to feet and legs until well warmed.

Quickly give vigorous cold mitten friction using ice water.

Rub dry with Turkish towel. Wrap well in warm blankets. Duplicate this treatment to thighs.

Then to arms, finally leaving patient warmly covered.

TREATMENT FOR COLLAPSE, which in this discussion means prostration due to primary circulatory inefficiency or to exhaustion, must be adapted to the systemic condition reflected by the patient's condition. The following measures are to be used, as they are indicated:

Relief of distress and anxiety, with morphine if severe.

Position of patient's body to favor cerebral blood supply, slight lowering of head with decided elevation of the legs by supporting them raised even to right angle with the body. Too much tilting of the thorax will interfere with filling of the heart.

Absolute quiet, to protect circulatory system from unnecessary demands, particularly needed in case of exhaustion or toxic paralysis of circulatory centers or muscles. Ice-bag on precordia to slow heart, should be removed and skin warmed by friction every four or five minutes.

Intravenous infusion, slowly but promptly given, if there has been loss of fluid from body by vomiting, purging, or hemorrhage. Use this measure with moderation in cases where there has been acting any drug that poisons the heart muscle or the inhibitor mechanism of the heart. Best solution, to litre of sterile, distilled water 9.0 g NaCl, 0.3 g KCl, 0.1 g CaCl₂, and 0.1 g Na₂CO₃ (Burroughs Wellcome & Co., Saline Compound No. 2, Soloid.) To this may be added 1.0 cc. Adrenalin chlorid solution, 1:1000. If adrenalin is added the pulse must be watched and the rate of administration slowed to a few drops per minute when it reaches a good condition. If much fluid has been lost so that the quantity to be introduced is larger, the adrenalin must be used more dilute in the first portions administered, or even withheld until it has been found that the pressure will not rise without it. If the heart muscle has suffered much one pint (500 cc) is the maximum quantity safely administered unless there has been great fluid loss. Simple normal saline, may be infused if nothing better available, but it is more likely to be followed by rigors, edema of the lungs, and other disturbances, due to unbalanced action of sodium ion. Adrenalin absolutely contraindicated after chloroform or other direct heart heart poison.

Hypodermoclysis or proctoclysis of value in less urgent cases, or where there is continuing loss of body fluids. Adrenalin by these routes of no value.

Bandaging of limbs and abdomen. A large area bandaged with moderate firmness gives more benefit than smaller area too tightly bound. For abdomen a many-tailed bandage best.

Pituitrin, slowly, intravenously may be given to raise blood pressure; but effect lasts for not more than about 20 minutes. Repeating dose soon results in production of vaso-

dilatation instead of constriction. Should never be given hypodermically in patient with very low pulse. A number of deaths are on record where stimulant drugs administered during shock and collapse remained unabsorbed until period of return of normal pressure, when rapid absorption of excessive quantities caused death of patient.

Maintenance of body warmth by hot blankets, hot water bottles, hot drinks, etc. Care needed to avoid burning patient, and to avoid weighting down chest with heavy coverings so as to impede respiratory movements.

Maintenance of respiration by artificial methods. When patient unconscious and exhausted the intratracheal insufflation method would probably be preferable to other methods where the apparatus is available. Osteopathic manipulations gently applied to upper dorsal and lower cervical regions has been seen to ease oppression of respiratory movements.

Administration of oxygen containing 3% to 5% of carbon dioxid, oxygen rendering a lesser degree of pulmonary ventilation sufficient to oxygenate the blood, carbon dioxid to overcome acapnia. Carbon dioxid would not be indicated if there is a tendency to lividity.

Stimulation is permissible when collapse is due to oligemia or oligemia combined with pain. It is **contraindicated when due to exhaustion** of circulatory organs. Best methods: **gentle osteopathic manipulations** as indicated, and **cold towel rub or bare hand friction** of the well-warmed skin.

Caffein, 1 gr (0.05 g); citrated caffein, 2 gr (0.1 g); or a cup of clear coffee, given by mouth; or a hypodermic injection of caffein and sodium benzoate. These may assist return of cerebral and medullary sensitiveness. Use more logical when there is not exhaustion of any form, but rather a simple depression.

Protection of a weakened heart muscle, while permitting it to do just enough work to keep blood circulating in vital centers, is the most important aim in the treatment of collapse with exhaustion.

A study of the treatments of shock, collapse and cardiac failure, as previously outlined, will reveal the following essential differences.

In shock,—after control of the pain, there is given vigorous stimulation of large cutaneous areas, including the thoracic wall, for the purpose of exciting restoration of spinal and cerebral tone, whose loss is considered the underlying cause of circulatory deficiency.

In collapse,—protection or assistance is given to the circulatory system according to its differing needs: protection, with avoidance of stimulation when there is exhaustion; assistance in every case where needed, with moderate degrees only of stimulation when there is no exhaustion.

In cardiac failure, before exhaustion stage,—protection from unnecessary functional demands, by repose accompanied by peripheral vasodilatation secured thru moderate cutaneous stimulation, excluding the thoracic walls.

Only rational interpretation of keen observations of exact circulatory condition can make possible the proper management of these conditions. Character of heart beat, tonicity of arterial wall, force and size and quality of pulse must be noted and compared with cerebral condition and evidences of spinal tone or atony.

In explanation of the policy of not recommending the usual stimulant drugs advised for use in "shock and collapse" the writer quotes the following:

"Personally, I do not hope for much from any of these drugs in shock." (Short, speaking of alcohol, strychnin, adrenalin, pituitrin, digitalin.)

"It is obvious that the use of stimulants of any sort is absolutely contraindicated, and that no more certain means of destroying any chance of recovery could be devised than the administration of strychnin." (Mummery, concerning shock, which he considers a pure case of exhaustion of the vasomotor center from continued attempt to respond to stimuli due to trauma.)

"Anything which will prevent the senseless administration of enormous doses of the so-called cardiac and arterial stimulants and tonics to patients who are in shock will redound to the advantage of the sick." (Handbook of Therapy, American Medical Association.)

"The more we know of shock and collapse, the less we pin our faith on drugs. If we employ them, we must not let the stress of the emergency lead us into giving them in too large doses." (Bastedo.)

"The more profound the shock, the more marked was the depressing effect of alcohol. * * *

"On the whole, nitroglycerine and amyl nitrite increased shock. * * *

"It seemed on the average, that cases of shock treated with digitalis did not live as long as the controls. * * *

"In shock therapeutic doses of strychnine are inert, physiologic doses dangerous or fatal. * * *

"It then follows that the treatment of shock by vasomotor stimulants in the form of drugs, is on precisely the same basis as treatment by burning the animal or crushing his paws. * * * It would seem to be as reasonable to treat strychnin shock by administering traumatism as traumatism by strychnin." (Crile, in "Blood Pressure in Surgery.")

"The success attained by the use of hydrotherapy has, in our hands, been uniformly gratifying. This has also been the experience of many others working in the association of medical institutions with which the writer is connected." (Abbott, Hydrotherapy.)

It is not possible to more than outline the reasons for regarding shock as being essentially a condition of loss of spinal and cerebral tone, accompanied by circulatory inefficiency. It is well at the outset to refer to the likelihood of the existence of two distinct conditions found, in these cases, with reference to the essential condition of the circulatory centers: (1) true exhaustion, found in those cases, particularly accompanied by somatic injury, where an initial overactivity has taken place; (2) reflex inhibition where visceral irritation has resulted in immediate fall of pressure with no period of overactivity of vasoconstrictor center, during which exhaustion might develop. It is also to be noted that there are two distinct modes of enlargement of capacity of the vascular system, namely, distention of arterial and distention of venous sides of the system, the latter being rendered possible by relaxation of the skeletal mus-

cles without any essential depression of venous tone. Finally, there are two modes of origin of loss of spinal tone: (1) cessation of tonic impulses to spinal centers from medullary centers; (2) excessive augmentation of inhibitory influences from the mid-brain or basal ganglionic centers. Particularly in case of deficiency of tonic descending impulses, it should be remembered that the cord possesses more or less ability to develop a tone of its own, dependent upon reflexes more nearly segmental in their activities.

Following rather closely the line of argument of A. Rendle Short, and being, perhaps, rather dogmatic for the sake of making clear in as small a space as possible one fairly definite concept of the nature of shock, we present the following argument in justification of the definition of the term which has already been presented.

“We have to find an explanation for the feeble, quick pulse, the falling blood pressure, and the mental and physical prostration.” It seems almost certain that **in shock there is not a condition of primary cardiac failure**; for, altho there is a diminished output from the heart, numerous observers find experimentally, in animals in this condition, a heart still capable of doing vigorous work, **but** failing to be efficient because of imperfect relaxation during diastole and of **poor filling of the ventricles**. Tho this impeded filling of the ventricles and consequent diminution of minute-volume of output may be in part due to some abnormal impulses from some of the circulatory centers causing increased tone of the ventricular wall (an action whose possibility is in doubt) it seems to be more directly **due to low pressure in the venae cavae**. (Crile, Henderson, Howell; cited by Wiggers.)

This low caval pressure could be due either to diminished volume of blood in the vascular system, or to dilatation and increased capacity of the vascular system for fluid. Malcolm believes that the constrict-

tion of the vessels, due to extreme pain, causes loss of fluid from the circulating mass and therefore an oligemia. Henderson believes that a condition of acapnia, developing, so alters the osmotic balance between the blood and the tissues that there is diversion of fluid into the tissues. Either of these processes would develop a definite increase in the specific gravity of the blood. This, however, fails to take place in many cases of shock (Short), the only typical rise taking place in case of actual loss of fluid from the system as by vomiting, purging, exudation from an extensive burn. On the contrary, in many cases there is a decrease in the specific gravity of the blood, indicating a movement of fluid from tissue to blood as evidence of an appreciation of the body for a need of increased blood-volume due to some increase in the capacity of the vascular bed—the same sort of a protective reaction as appears in hemorrhage. (Adami, Pathology.) It seems certain then that the low pressure in the venae cavae is **not essentially due to a total oligemia.**

In the vascular bed, the arterial and the venous sides are each capable of considerable and independent variations in capacity. Accumulation of blood in one or the other or both of these areas might account for the slow flow and low pressure in the cavae. That it is **not due to augmented capacity of the arterial bed** in true shock is indicated by many experiments and observations (tho such may be the case in the more slowly developing exhaustions of collapse). In fact, perfusion experiments, upon kidneys, intestines, and limbs of animals in shock, indicate an active discharge of vasoconstrictor impulses after shock has supervened and blood pressure fallen, just as in a fall of blood pressure from other cause. (Wiggers, and others.) After death from shock there is usually constriction, not dilatation of the arteries. “The skin is pale, the pulse is small, bleeding is scanty, there is anuria (suggesting renal anemia), the retinal vessels are contracted.” (Short.) “It would seem,

therefore, that dilation of the arterioles is neither responsible for the decreased return of blood to the heart, nor for the fall of arterial pressure." (Wiggers.) It seems that there can be **no essential vaso-motor center exhaustion**, tho this condition could conceivably develop as an effort of the center to raise a pressure which has been lowered by some antecedent influence. (In the terminology of this discussion, therefore, it is possible that a **long-continued condition of shock would pass into one so nearly resembling collapse as to render inadvisable the treatment given for early shock.**) In this manner can be explained the occasional, but not regular, findings of histologic evidence of exhaustion of medullary centers, assumed by such workers as Mummery to be the vaso-motor centers.

At least one worker has secured definite results that strongly indicate that there is **no accumulation of blood in the abdominal organs**, as is so widely taught. **But there is accumulation of blood in the large veins of the abdomen and the deep muscle-supported veins of the limbs**, while the subcutaneous veins are drained empty.

Altho the "acapnia theory" of Henderson and the "adrenalin exhaustion theory" were especially advanced to explain the supposed accumulation of the blood in the abdominal organs, the venules or the arterioles, which seem from the preceding views to have relatively little to do with the essential origin of shock, it is worth the space to mention that neither of them is supported by the extensive observations made by Short upon patients and at post-mortems. However, the acapnia theory would so nicely explain some of the indications of lessened sensitiveness of the medullary centers, such as loss of tone of the respiratory center, that one is reluctant to abandon it; and there is some clinical evidence that seems to justify admixture of carbon dioxid to the extent of about 5% with oxygen for respiration in these cases. No discussion of this question can be attempted here

because of the many considerations involved. We can simply cite that Short did not find the amount of carbon dioxid in the blood of patients, to be below normal. But it might be well that, in this condition of depression characterizing shock, the body could utilize a greater than normal proportion of the hormone-like substance.

Short's view of the origin of the venous stasis, which seems to be the essential circulatory phenomenon of shock, is presented thus:

“When the voluntary muscles are paralysed, as by curare, blood-pressure falls, altho curare does not act on nerve-endings in involuntary muscle. The reason is that **by loss of muscular tone the veins lose an important support, dilate, and become reservoirs of more or less stagnant blood.** * * *

“In my opinion the cause of surgical shock is paralysis of the great nerve-cell groups about the fourth ventricle, including, it may be, the vasomotor center, leading to mental and physical prostration, tonelessness, and, **owing to the atony of the voluntary muscles, pooling of the blood in the great deep veins,** inefficient filling of the right heart, reduced output by the left ventricle, and consequently constricted arteries and eventually a low blood-pressure or sudden death. Thus can we explain the low blood-pressure in spite of strong heart, contracted arteries, and normal blood volume. Here also we find an explanation for the absent knee-jerks and other reflexes in severe shock—they are absent just as they would be in spinal shock, from failure of the impulses descending from the great nerve centers of the fourth ventricle.”

The present writer ventures to criticise Short's explanation of the manner of production of loss of spinal tone, in that there is no recognition made of the probable existence of great inhibitory tracts being distributed, not only to the medullary centers, but to the spinal muscle centers at all levels of the cord; that over-activity of the centers giving origin to

these tracts in the mid-brain region might readily produce such profound relaxation as observed in shock, at the same time depressing cerebral tone,—both phenomena which are recognized as occurring often before or simultaneous with lowering of blood-pressure; and finally, that the data cited by Short in explaining experimental “spinal shock” seem just as capable of explanation in terms of the physiology of descending inhibition tracts, as by assumption of the loss of tonic impulses. In fact, the indications are that normally both types of impulses affect the spinal centers. But the rapid restoration of spinal tone by the hydrotherapeutic measures of Abbott, and the rapid disappearance, following osteopathic manipulations and reassuring suggestion, of the symptoms of shock in some of the writer’s cases, lead him to regard the inhibition from the mid-brain as being an important factor in the induction of shock. This view of its origin also **accounts for the early appearance of mental prostration**, even before the development of circulatory inefficiency, which is not explained by Short’s statement, in spite of his emphasis upon **the fact that shock may commence long before the pulse weakens and becomes rapid.**

The rapid progression of the condition, when profound, to one involving true exhaustion of certain medullary and, possibly, mid-brain centers seems only too well proven. Hence the necessity of prompt application of measures to restore spinal tone,—measures well illustrated by the vigorous rubbing of the skin in cases of syncope which can be regarded as that mild degree of shock from which there is a tendency toward spontaneous recovery.

Tho the foregoing is but an attempt to hurriedly correlate the indications of the various investigations on this subject and can make no pretense of thoroughness nor conclusiveness, it is hoped that analyzing the argument will assist the reader to a recognition of the varied factors and manifestations of these conditions, or bestir him to form for himself a clearer conception of their underlying nature.

THE SPECIAL MANAGEMENT OF INDIVIDUAL CASES OF POISONING.

The diagnosis of the particular poison active in any case is essential to the most successful management of the case, tho many cases may be managed quite as well by the application of the foregoing general principles. In some cases the exact diagnosis is indispensable. Making the diagnosis may be very simple or may be practically impossible, according to the nature of the poison and the possibility of securing an intelligent history of the case. In determining between an acute illness and acute poisoning, there will frequently be observed all that is requisite for this specific diagnosis. The following points may be kept in mind during the efforts at diagnosis:

Attempt to put the case into one of the general groups, I, II, III, or IV.

- I. Gastric or abdominal pain; vomiting; shock, rapid weak pulse.**
- II. Cerebral excitement; abnormal muscular activities; pulse, rapid but full.**
- III. Physical prostration from either drowsiness, peripheral paralysis with senses acute, or circulatory paralysis not due to shock.**
- IV. Primary disturbances of respiration, as asphyxia and irritation of respiratory tract.**

Recall the specific symptoms of the common drugs in group to which the case belongs and look for those symptoms.

If believed to be suicidal attempt recall and look for symptoms of commonly used suicidal drugs.

Do not delay in order to make a specific diagnosis before emptying the stomach. This procedure should be done as soon as poisoning is recognized as the cause of the patient's condition. **Wait only long enough to decide upon wisest method to use.** Continue efforts at diagnosis while carrying out this procedure.

When any drug is suspected, recall lapse of time required for symptoms to show themselves, and determine whether patient has ingested anything, or otherwise been exposed to possibility of poisoning, within the corresponding period. (There are relatively few gastro-intestinal irritants, for example, with which more than half an hour could elapse before onset of some of the symptoms.)

In examining the patient make note of:

Breath,—odor.

Buccal Mucous Membranes,—swelling, corrosion, discoloration, dryness.

Vomit, —odor, chemical alteration, color, fresh blood, altered blood, nature of food particles, remnants of poison.

Condition of Muscular System,—flaccid, normal tone, excessive reflexes, loss of reflexes, twitchings, cramps, clonic contractions, tonic contractions, paralysis.

Mental State,—unconscious, stuporous, comatose, recurring syncope, legarthy, hallucinations, delirium, consciousness unusually clear.

Compare particularly with physical condition.

Pulse,—rate, slow, fast or irregular; weak, small or full; soft; firm; wiry.

Skin,—warm, cold, dry, moist, perspiring, red, pale, cyanotic, icteric, eruption.

Pupils of Eye,—dilated, constricted, non-responsive to light.

Pain,—colicky, burning, in muscles, mouth or throat, other location.

Genito-urinary Organs,—strangury, priapism, abortion, suppression of urine.

Urine,—color, odor, blood, methemoglobin,, bile, quantity, reducing power.

Stools,—frequency, consistency, odor, fresh blood, altered blood.

Temperature (by thermometer).

Visual Disturbances,—amaurosis, diplopia, colored images.

From the various findings attempt to recognize the probable internal actions of the poison to produce the particular complex found, and from knowledge of pharmacology attempt to select drug causing such an effect.

GROUP I.

Gastro-Intestinal Irritation.

(Individual Poisons Arranged Alphabetically.)

Immediate Symptoms:—Abdominal Pain,

Vomiting,

Purging, within a short period.

Shock and Collapse.

(Some members of the group in dilute solutions fail to produce these symptoms, becoming specific irritants.)

After Symptoms:—Inflammation or ulceration of mouth, throat, stomach or intestines.

Jaundice,

Peritonitis,

Nephritis.

Remote Symptoms:—Strictures of alimentary tract structures,

Atrophic gastritis,

Inanition.

General Treatment:—Antidotal. (Stomach usually spontaneously emptied.)

Demulcent.

Pain Control,

Management of shock, and other symptoms.

“ACIDS.”

Corrosive Poisoning, caused by all

Strong Mineral Acids,

Sulphuric Acid, or Oil of Vitriol (corrosion white changing to brown);

Hydrochloric Acid, or Muriatic Acid (corrosion greyish-white);

Nitric Acid, or Aqua Fortis (corrosion bright yellow);

Zinc Chlorid, or Soldering Fluid (contains HCl).

Oxalic Acid, pain develops more gradually (see “Oxalic Acid”);

Carbolic Acid, recognized by odor (see “Carbolic Acid”); and

Alkalies, with which the corrosion is soft and slimy, not tough or brittle as with acids.

Source.—Sulphuric, Hydrochloric and Nitric Acids can be considered together for clinical purposes, as their effects are essentially the same. As a class, they constitute a fairly frequent cause of poisoning. Altho all of these acids are widely used in the arts and trades, about half of all of the cases are suicidal attempts. The accidental cases seem more frequent among children. Sulfuric about twice as frequent as the other two combined.

Fatal Dose.—Difficult to state as adults have been killed by 1 dram, and have made partial recoveries from as much as 70 drams. Death likely to follow dose of $\frac{1}{2}$ ounce. When taken with suicidal intent mortality ranges from 65% to 85%.

Action.—Prompt local destruction of tissue, depending upon concentration of ingested drug and duration of action. Injury likely to produce shock. Irritation to stomach causing severe reflex vomiting, tends to bring on collapse and exhaustion. Acidosis apt to result from absorption, and may result in death during period of a few days. Late death from destruction of alimentary mucous membranes. One-half of deaths during first 24 hours from shock and collapse. One-half of remaining deaths occur during the first week from exhaustion or acidosis.

Symptoms.—Instant agonizing pain as soon as parts are touched by acid.

Swelling of tongue and mucous membranes, with whitening at first.

Salivation, with inability to swallow because of pain.

Intense thirst.

Vomiting and retching, persistent.

Vomit contains acid, much mucous, shreds of mucous membrane from esophagus and stomach, and blood.

Shock and Collapse.

Sometimes **asphyxia**, from corrosion and spasmodic closure of the glottis.

If recovery from collapse, there may develop within several hours or several days, stupor or convulsions indicating acidosis.

Intense gastro-enteritis with possibility of rupture or perforation of the viscera must be anticipated as possibilities.

After symptoms and remote symptoms typical of this group are of common occurrence.

Immediate Treatment.—

Give freely of milk containing 4 oz. milk of magnesia to the quart. (Or, milk or water containing powdered chalk, magnesium oxide, sodium bicarbonate, or soap solution. The carbonates are not entirely free from the danger of severely distending and contributing to perforation of the stomach.)

If there is nothing else at hand, at once give large draughts of milk or water, to dilute the acid and check its corrosive action. The antidote may be given when it is secured.

There is seldom any need for an emetic, vomiting is likely to be too profuse without it. A few workers recommend lavage, but the tube may easily perforate the corroded stomach or esophageal wall, so the process is dangerous. Altho it is fatiguing, the vomiting of several successive draughts of milk containing an antidote accomplishes the same purpose. Where corrosion is slight, acidosis may be prevented by lavage.

Control pain with demulcents, chipped ice, or other measures.

Shock and collapse should be treated promptly, continuing as much stimulation as the degree of collapse permits without augmenting the exhaustion.

Asphyxia, from edema or spasm of glottis, may require immediate **tracheotomy.**

After Treatment.—

Anticipate and treat acidosis.

Surgical management of gastro-intestinal corrosions. Skiagraph of value in determination of severity of condition.

Supervision of feeding.

Enforcement of **prolonged rest.**

Sequelae.—Strictures at various points of alimentary canal.
Atrophy of gastric mucosa.

“ALKALIES.”

Caustic Alkalies, all produce Corrosive Poisoning,—

Caustic Soda, or Sodium Hydroxide;

Caustic Potash, or Potassium Hydroxide;

Washing Soda, or Sodium Carbonate;

Lye, or Soda Ash;

Ammonia Water, or Ammonium Hydroxide (which see).

Source.—Common household substances for cleansing purposes, so easily secured accidentally by children. Occasionally used suicidally.

Fatal Dose.—One-half ounce of strong solution, tho more may be recovered from. Mortality from 65% to 75%.

Action.—Solvent action upon tissues, with no tendency to coagulate as acids possess. **Corrosion soft and extends deeply.** Lack of astringent tendency causes freer passage of poison into intestines than with acids; so **intestinal symptoms, both immediate and remote, are increased.** Early death from shock or collapse in 3 to 24 hours. Death from inanition months afterward is of frequent occurrence.

Symptoms.—Soapy taste followed by burning.

Marked abdominal pain soon develops and becomes aggravated.

Colic and purging in an hour or less, if poison enters intestine.

Stools and **Vomit** contain blood and mucous membrane shreds; vomit may be slimy and **will be alkaline (red litmus turned blue).**

Corrosion, differs in appearance from that of acid, the latter being brittle instead of slimy.

May be shock; later profound exhaustion from purging.

Remote symptoms as for acids, but no acidosis.

Treatment.—Emetic and Stomach-tube not permitted.

Give large draughts of vinegar in water (tumbler full to quart).

Or, better, citric acid, tablespoonful dissolved in quart warm water.

Or, strong lemonade. **Continue until vomit contains an excess of acid.**

Later, give large quantities of milk or of olive oil, and continue as long as any demulcent effect can be observed.

Control pain, shock, and collapse.

Flushing colon may help control purging. Use water at 102° F.

Asphyxia may call for tracheotomy.

Manage late symptoms as indicated. **Rectal feeding** almost certainly necessary for a considerable period.

Remarks.—It is very important to be able to distinguish this type of poisoning from that of the strong acids, as antidotes are an important part of the treatment and must be specifically chosen.

Ammonium Hydroxide, see Alkalies.

This is less corrosive than the other corrosive alkalies, but more likely to cause edema of the glottis, because of its volatility, and sometimes is rapidly absorbed, causing sudden appearance of delirium, coma, arrest of the heart and death inside of a few minutes. Fatal dose, about the same as preceding. Symptoms, the same as for alkalies with the addition of those from systemic action. **Treatment**, see Alkalies.

Antimony, usually as

Tartar Emetic, or Potassium Antimonyl Tartrate.

(Antimony Trichlorid, rare.)

Source.—Usually antimony poisoning results from overdose of tartar emetic, or from taking this drug in place of some other medicine. Extremely rare suicidal poison; more frequently homicidal.

Antimony trichlorid is extremely rare as a cause of poisoning, being a rather typical corrosive when it is.

Fatal Dose.—Difficult to state. Many deaths from less than 10 grains in divided doses; many recoveries from single doses of 30 grains or more. 15 to 20 grains probably most dangerous size for single dose. Mortality, uncertain; probably not more than 40%.

Action.—Tartar emetic is not corrosive, but a protoplasmic poison. Action slow in appearance because metal must enter cells before injuring them, setting up sharp inflammatory reaction. Doses which are not emetic more apt to reach the intestines, to be absorbed and thus extend toxic influence. After absorption irritates, then depresses heart, nervous system and other portions of system. Death from cardiac paralysis augmented by collapse.

Symptoms.—Also extremely variable. Most nearly typical are

Metallic taste;

Gastric pain, appearing between fifteen minutes and one hour after dose;

Nausea and Vomiting, usually profuse;

Purging, stools "rice-water" type, accompanied by tenesmus;

Collapse, with frequent fainting.

After absorption,—

Muscular tremors, cramps in limbs;

Delirium;

Cardiac failure (collapse type) from direct toxic action.

Treatment.—Administer promptly and freely **strong tea or coffee**, for the precipitant action of the tannic acid upon antimony (or use tannic acid, 60 gr. to pint of water).

Wash out stomach with coffee or tea if that given by mouth has not been vomited back. Then,

Use demulcents and other measure to **control pain** and vomiting.

Manage Cardiac Failure and Collapse, remembering replacement of body fluid lost.

Remarks.—No method of checking action upon vital tissues after absorption, therefore cases where vomiting is absent or much delayed are most often fatal.

Diagnosis is important and may be difficult. Information as to presence in house of tartar emetic or arsenic, of history of patient having just taken a dose of medicine and nature of illness for which taken, might give clues. **A test of the suspected material** may be made quickly, if reagents available: small quantity dissolved in water, acidified with HCl, add two drops Ammonium sulphid or better several drops of hydrogen sulphid water. Precipitate from antimony is bright orange-red; from arsenic would be bright yellow.

ARSENIC.

Arsenic.

White Arsenic, or Arsenic Trioxid;

Paris Green, or Aceto-Asenate or Copper;

Poison Fly Paper (element or oxid);

Insect Powders (many contain one of preceding);

“Rough on Rats” (may contain either As or P);

Fowler’s Solution (potassium arsenite);

Donovan’s Solution (AsI₃ and HgI₂ in solution).

Source.—Poisoning has occurred in the most varied manners: use in cooking in mistake for baking powder, drinking beer from glucose made with impure sulphuric acid, administration for skiagraphic purposes of impure bismuth subcarbonate containing arsenic, suckling infant by mother who was being criminally poisoned by small doses, as well as from eating of insect poisons and fly paper by children, and the numerous suicidal and homicidal and medical over-dosage cases. Lay use of the drug in skin lotions and as abortifacient has caused death. Because of high frequency of employment for criminal poisoning, and former frequency of its suicidal use, together with the ease of obtaining it, many toxicologists consider arsenic the most important poison.

Fatal Dose.—3 grains, if absorbed. Dangerousness and symptoms both vary greatly with different conditions. A concentrated, readily absorbed dose might cause death in few hours by systemic action; a concentrated but difficultly absorbed dose might emphasize tendency to delayed collapse due to extension of action thruout the intestinal area but excretion keeping pace with absorption so as to prevent action upon the central nervous system. Even knowing the conditions attending the administration of the drug it is difficult to foresee the exact mode of action. A few hours to many days may be occupied in causing death.

Action.—Arsenic is a true protoplasmic irritant; non-corrosive, but exciting inflammation and degeneration of the cells it may enter, thus causing, for example, death and permitting erosion of the gastro-intestinal mucous membranes. After absorption, it produces primary irritation and subsequent progressive degeneration of the functioning cells of the body organs, probably due to interfering with normal cellular oxidations. The endothelium of the vascular system particularly suffers, including that of the heart. Symptoms are due to the gastro-intestinal inflammation, its resultant prostration, and the later systemic poisoning. Tho almost identical with antimony in mode of action, it is much more readily absorbed by the cells of the body, and therefore a more vigorous poison.

Symptoms.—As with antimony, these vary in different cases
There are **three general types** in the progression
of the symptoms:

Gastro-enteric type,—

Burning pain, develops in stomach, esophagus and throat about 15 minutes to one hour after injection, and **increases on pressure** over epigastrium, because of inflammatory origin;

Great thirst, from inflammatory reaction in throat and loss of body fluids;

Nausea, and

Vomiting of bile-stained stomach contents, intensity creasing with time, instead of diminishing, until exhaustion ensues;

Colic, as inflammation comes to include intestines;

Diarrheal, "rice-water" stools, of serous exudate with rolled-up flakes of sloughed mucous membrane, sometimes blood-stained;

Tenesmus; all these co-operating to induce exhaustion, which is complicated by appearance of **systemic effects**;

Muscular cramps, or mild convulsive spasms of the limbs;

Delirium, finally followed by profound

Collapse, and death inside of 30 hours.

Narcotic type is rare. Here is observed an almost selective action upon the central nervous system with the preceding symptoms absent or mild. A somnolent condition develops an hour or so after injection of drug, delirium interrupts the depression, after which the patient becomes

Comatose and dies, often without gaining consciousness, in about 8 hours.

Mixed type,—usually duplicates the foregoing

Gastro-intestinal irritation symptoms, followed by

Partial recovery within 24 or 36 hours. But

Suppression of urine becomes more marked from acute glomerular nephritis,

Exanthemata of varied nature appear,

Edema of the face or icterus may develop,

Headache,

Delirium,

Progressive weakening of each vital function is observed,

Coma, sometimes interrupted by convulsions, followed by

Death in from 3 to 10 days.

Treatment.—Antidotal treatment important, even if some time has elapsed since onset of symptoms, because form of poison often such that it adheres to the gastric mucous membrane to be slowly absorbed. This same fact makes it difficult to make sure of removing the poison by treatment. If there is knowledge of the taking of a dose of arsenic,

Empty the stomach without waiting for vomiting. An emetic is preferable in case there is food in the stomach.

Give antidote,—

(1) **Ferric Hydroride with Magnesium Oxid, prepared extemporaneously:**

(a) Put 2 teaspoonfuls of Magnesia in a tumbler, add 2 teaspoonfuls of water and stir to a paste; fill glass two-thirds full of water and mix; then pour in with stirring $\frac{1}{2}$ ounce of tincture of ferric chlorid. This makes one dose. **Give several doses, 10 minutes apart.**

or (b) Put 3 ounces of tincture of ferric chlorid into a pint of water; pour in ammonia water with constant stirring until the well-mixed mass smells slightly of ammonia. Then strain the mixture thru a couple of thicknesses of muslin, pour water over it and let it drain twice. **Give 2 teaspoonfuls of the brown jelly-like mass every 10 minutes.**

or (2) **Dialysed Iron**, one teaspoonful, followed by teaspoonful of table salt dissolved in water. **Repeat dose every 5 or 10 minutes for eight doses.**

Use lavage if stomach has not emptied itself during the administration of the antidote.

Pain may be controlled by hot abdominal fomentations or otherwise.

Give freely of milk containing $\frac{1}{2}$ teaspoon of sodium bicarbonate to the tumbler full.

Control collapse, remembering likelihood of need to replace lost body-fluid with venous infusions. The infusion solution should contain sodium bicarbonate, 30 grains to the pint, which seems to check degenerations.

A medium dose of castor oil to **prevent closing up of bowel** too early is recommended, followed by liberal doses of milk, olive oil or other demulcents.

Sequelae.—Gastro-enteritis, sometimes of atrophic type; nephritis; myocarditis; “neurasthenia”; anemia.

Diagnosis.—Where the symptoms have occurred without any history of the taking of the drug “the autopsy with the chemical analysis of the organs will remove all doubt.” The vomitus may contain some of the substance which was taken which may be recognized. The pain in the throat would likely not be present in many forms of acute enteritis; in peritonitis the localization of the pain and lack of the sharp burning nature would be points of difference. Finding the arsenic in the urine would be absolute proof: acidify with HCl, and subject to Marsh’s Test.

Barium Compounds.

Barium Chlorid;
Barium Sulphid, used in depilatory lotions;
Barium Carbonate.

These are but rarely causes of poisoning. Only the sulphid is corrosive; the chlorid in strong solution irritant; carbonate no local action. After absorption there is marked contraction of all smooth muscles of the body, resulting in elevated blood-pressure. Limbs, tongue and pharyngeal muscles tend to be “stiffened.” Hallucinations appear. Later convulsions, paralysis, collapse. These are added to gastro-intestinal irritation.

Treatment.—Lavage with solution of sodium sulphate, 1 teaspoonful to the pint of warm water. Nitroglycerin, 1/200 grain repeated three or four times at 15-minute intervals, to lower pressure arterial. Manage pain and collapse.

CARBOLIC ACID, or Phenol.

Cresol, Creolin,
Lysol, and other relatives cause same condition.

Source.—Wide use as antiseptic results in its being taken by mistake as a colored medicine or as castor oil, many of the deaths even being due to the errors of nurses in administering it. Numerous suicidal cases. Several deaths on record due to its absorption from surgical dressings or from douches. One of the most common poisons.

Fatal Dose.—Death likely to occur from swallowing one teaspoonful (5 gm.), but many recoveries have been secured from 10 times this quantity (50 gr., or 2 ozs.). Mortality about 50%.

Action.—Corrosive before absorption. Systemically, first irritates, the profoundly depresses nervous system (particularly the centers of the medulla) and the heart. Excreted partially as ethereal sulphates, partially as hydroquinol; the former replaces the mineral sulphates of the urine, the latter exposed to the air becomes black on oxidation.

Three modes of death—by shock from pain of corrosion, in 5 minutes to 2 hours; (2) by collapse from combined influence of corrosion followed by rapid absorption and systemic action, in 2 to 36 hours; (3) by collapse from depression after slow absorption of dilute form, in 6 hours to 3 days. Death may be longer delayed, particularly in second form—the most common of the three—from the typical late and remote effects of corrosive poisons.

Symptoms.—According to the three modes of action:

- (1) Immediate burning pain, in mouth and throat.

Shock, sometimes without vomiting.

Mucous membranes swollen and whitened, later becoming brown.

Odor of Carbolic Acid.

- (2) Burning, tingling, then numbness of mucous membranes.

Corrosion and odor as in first type.

Gastric Pain, and

Vomiting, vomit possessing odor of poison and often shreds of mucous membrane or blood.

Shock, changing to collapse with muscular tremors, and sometimes delirium, from systemic effect after rapid absorption induced by the corrosion. Pryexia sometimes set up during preliminary period of nervous irritation.

Urine will give no precipitate of sulphates with barium chlorid solution after first few hours, and will become dark on standing. May contain blood and albumin.

Profound respiratory depression and cardiac poisoning in critical stages.

(3) No corrosion. Breath may have odor.

Gradually developing and progressing collapse with all symptoms, several hours occupied by the process.

Sometimes suppression of urine with uremia.

Urine, no precipitate with barium chlorid; may darken on standing, and show albumin.

Treatment.—Administer several ounces of 1 part alcohol in 4 of water (or mixture of equal parts of whiskey and water); or

Promptly introduce the stomach-tube, using care not to introduce too far, and wash out the previously given antidote. Wash out the stomach, without removing the tube, with several more portions of the alcoholic solution, or with water if nothing else available, until the washings return free from carbolic acid. Olive oil may be used instead of alcohol, but it must be promptly removed. All of these antidotes act merely by dissolving the acid and assisting in extracting it to some extent from the stomach mucous membranes. They do not chemically neutralize it. Sodium sulphate has been proven not to be an antidote. **Emetics cannot be relied upon**, because of local anesthesia.

Give demulcents freely, but **PROHIBIT** use of **OILS**; egg white the best form, skimmed milk permissible.

Treat shock or collapse as indicated. In collapse mild stimulant measures are permissible to maintain spinal tone, but patient must be kept from putting any demand upon his heart because of the direct toxic action it is likely to have taken place.

Sodium sulphate to replace the sulphate carried out of the system, may be added to the saline infusion. 5 g. to the litre; not more than 5 g. used in 12 hours unless urine shows continued absence of sulphates.

Anticipate and treat suppression of urine.

Enforce a prolonged rest after immediate symptoms have been recovered from. After effects of corrosions managed as under "Acids."

Caustic Alkalies, see Alkalies.

Colchicum (Meadow Saffron).

A drug containing colchicine, an alkaloidal protoplasmic poison, widely used in the treatment of gout. Fatal dose, probably about 20 times the medicinal dose. A rare poison.

Symptoms.—Typically those of gastro-intestinal irritation group, but **non-corrosive** and **requiring a period of an hour or more for the development of severe symptoms.** Death from respiratory failure, in collapse, in about 8 to 24 hours after fatal dose. **Nephritis, Convulsions,** and sometimes an ascending paralysis are **superadded symptoms.** History of taking the drug would be necessary for diagnosis.

Treatment.—Wash out stomach thoroly with water, then with warm strong tea, or 1 dram tannic acid dissolved in pint of water. Flush out colon with tea or tannic acid solution.

Demulcents may be given but not much effect expected from them.

Treat collapse and suppression of urine.

Copper Sulphate, Blue Vitriol.

Source.—Usually administered either with homicidal intent or taken suicidally. Few cases of death on record.

Fatal Dose.—About 1 ounce, most vigorous action when in strong solution.

Action.—Irritant to gastro-intestinal tract, but non-corrosive. Probably some absorption, with some irritant action upon the liver and kidney. **Death usually from combined exhaustion, icterus and nephritis.**

Symptoms.—Complete set of symptoms of gastro-intestinal irritation, with rather slow onset, epigastric pain not occurring until many minutes after administration. **Metallic taste, blue or green discoloration of vomitus, dark feces, cramps in legs; urine scanty and containing altered hemoglobin, making it dark.** Collapse from exhaustion and not of intense degree.

Treatment.—Empty stomach, then wash it thoroly with 1% solution of potassium ferrocyanid, and finally with water. Treat symptomatically.

Cresol.

Effects similar to and treatment identical with that of **Carbolic Acid** when latter has been taken in relatively dilute form.

Croton Oil. Oleum Tiglii.

Causes occasional accidental deaths from overdoses. A powerful gastro-intestinal irritant showing all the typical symptoms in marked degree. Added to these are some signs of systemic action,—delirium, followed by respiratory failure. **Collapse profound.** Death usually in less than 12 hours. Fatal dose, 20 to 30 minims.

Treatment.—Lavage, demulcents, pain control, collapse management.

Digitalis.

Is rarely ever a poison, except in connection with medicinal over-dosage or cumulative effects. Other symptoms usually develop before the gastric irritation, which is of relatively little importance in the production of the patient's condition. See discussion under **Group III.**

Food Poisoning.

While it is true that there is a large number of annual deaths from so-called "food-poisoning," it is not possible to consider this subject in this manual, in more than a general way. Much of what is called "ptomain poisoning" is in reality **anaphylactic shock.** Most of the meat "poisonings" are in reality cases of infections with members of the paracolon group of bacilli, and therefore show a distinct tho short (18 to 48 hours) incubation period and a distinct febrile condition. Each of these will have certain characteristics which must be learned elsewhere. In any event of this nature, however, even tho the offending substance itself had passed on out of the stomach, no harm would be done and possibly much good by performing lavage and cleansing the bowel, and then following the symptomatic treatment indicated by the patient's condition. Certain rare types of **fish poisoning** might be tho of as true poisonings, but they do not occur in this part of the world to any extent. Mushroom poisoning, however, is a case of true poisoning, and is discussed under the head **Mushrooms.**

Formaldehyde Solution.

Formalin.

Formaldehyde Gas may be inhaled and therefore comes for consideration in **Group IV**.

Source.—Occasionally accidental ingestion, but more frequently taken suicidally. Number of cases increasing. Common use as disinfectant solution makes it easy to obtain.

Fatal Dose.—One-half ounce or more.

Action.—Locally destructive to tissues it reaches, with some absorption and systemic irritation.

Symptoms.—Typical of gastro-intestinal irritation, including diarrhea with tenesmus, and having added albuminuria, hemoglobinuria or anuria.

Irritation of vapor may effect eyes and nose.

Sometimes the vapor reaches the larynx in sufficient amount to produce edema of the glottis with asphyxia.

Treatment.—Ammonia is the chemical antidote but ignorance of amount and location of the poison makes it difficult to gauge its administration intelligently. Give about a pint of ammonia water so well diluted that its vapors can be readily inhaled without nasal irritation.

Then empty stomach, with stomach-tube if possible, and wash out stomach with more of the dilute ammonia.

Give egg white freely, or milk which acts as further antidote.

Treat pain and Collapse or Shock.

Anticipate Uremia and manage case correspondingly.

Tracheotomy or Intubation may be needed.

Sequelae.—Gastro-enteritis and nephritis.

Fungi, see Mushroom.

Hydrochloric Acid, see "ACIDS."

Lead Compounds.

(As an acute poison lead occupies an unimportant place nowadays. It is the most important of the chronic poisonings. See Chronic Poisoning.)

Sugar of Lead, Lead Acetate.

Goulard's Extract, Solution of Lead Subacetate.

White Lead.

Source.—Use as astringent wash or lotion permits its occasional accidental ingestion. White lead has been used as an abortifacient.

Fatal Dose.—Fatalities are rare; percentage of recoveries, high. Death not at all likely to occur from less than one ounce, and several recoveries are recorded after six ounces of white lead.

Action.—Locally precipitates protoplasm, causing gastro-intestinal irritation. **Absorption slow.** **Systemically, general protoplasmic irritation** of moderate degree; causes contraction of all smooth muscle and tends to interfere with relaxation of voluntary muscle, as well as mildly irritating widespread nerve tissues.

Symptoms.—**Sweet, metallic taste** (from acetate) persists often.

Gastric pain and Vomiting, usually in less than half-hour, if due to strong dose of acetate or subacetate.

Colicky pains, reduced by pressure, because of non-inflammatory origin.

Spasm of intestinal muscles tends to retard evacuation of bowel, but there is sometimes diarrhea. Stools black from PbS.

Muscular cramps, vertigo, headache, insomnia, appear during few hours.

Urine diminished. In fatal cases there is

Marked depression, coma and death in 2 to 3 days.

Treatment.—Administer 1 ounce of **Sodium or Magnesium Sulphate**, dissolved in water.

Remove with stomach tube or emetic, and if possible perform lavage with the antidote.

Give freely of egg white or milk.

When stomach quiet, give $\frac{1}{2}$ ounce Magnesium Sulphate in milk.

Treat symptoms. **Atropin may check colic**, $\frac{1}{100}$ gr. with morphin $\frac{1}{8}$ gr.

Sequelae.—Insomnia, nephritis, persistent constipation, neuritis.

Lye, see "ALKALIES."

Lysol, see Carbolic Acid.

Number of cases from this substance is increasing, due to growing employment as an antiseptic solution. There have been a number of cases develop from absorption from surgical dressings and vaginal douches. When taken in strong solution by mouth, **resembles Carbolic Acid, Group I**; when from surgical dressings, Carbolic Acid, Group III.

Matches, see Phosphorus.

MERCURY.

Corrosive Sublimate, Mercuric Chlorid,
"Bi-Chloride";

Antiseptic Tablets, or Bed-Bug Poison;

White Precipitate, or Ammoniated Mercury;

Red Precipitate, or Mercuric Oxid;

Calomel, or Mercurous Chlorid; and

All other compounds of Mercury are poisonous.

Source.—The widespread use of corrosive sublimate as an antiseptic, of calomel as an internal medicine, the ease of securing these substances, popular knowledge of their employment and toxicity of small quantities after absorption, combine to make mercury one of the most important of modern poisons. The frequency of mercury poisoning has increased during the past few years, so that precautionary measures are being taken to diminish the likelihood of accidental poisoning. The numbers of suicidal and accidental cases, at least up to 1910, were about equally proportioned. The accidental poisoning is most frequently because of mistaking the bichloride tablets for other medicines. **Numerous other accidents in connection with therapeutic uses occur, however, as, poisoning from retention of antiseptic douches, from absorption from ointments or surgical dressings, from over-dosage of calomel, etc.**

Fatal Dose.—3 to 5 grains in form to be rapidly absorbed; much less seems to have caused death in connection with therapeutic over-dosage; 2 grains of calomel repeated for 10 days has caused death; 100 grains of corrosive sublimate has been recovered from when treatment followed promptly after the ingestion of the drug. Mortality 50%.

Action.—Locally in strong solution is corrosive. After absorption the metal is widely carried thruout the body tissues, exerting a protoplasmic irritant effect wherever it goes. Its rapid diversion to the eliminating organs, the kidneys, colon and other alimentary tract mucous membranes, results in the development in these tissues of inflammation corresponding to the amount of the drug reaching them. At the same time this tends to diminish the irritation of the other tissues of the body, tho the irritation does actually develop even in the tissues of the central nervous system where it produces changes such as found in acute myelitis.

Symptoms.—Besides the chronic type (which see), mercury poisoning is seen in several acute forms. (1) When taken by mouth in concentrated solution of the bichlorid, there may be death from shock or collapse after a short (few hours) duration of symptoms typical of the corrosive poisons, but a little less intense. Even in this form there is likely suppression of urine. (2) When taken by mouth in fairly dilute solution, or when taken as a tablet undissolved, we tend to have what will be described as the usual form. (3) When due to a slowly absorbed form or from absorption thru the skin or genital mucous membranes, the gastro-intestinal symptoms appear about the same time as suppression of urine, and commence with salivation and colitis rather than gastritis, resembling strongly the symptoms of the 3rd period of the typical form of mercury poisoning.

1st Period,—of usual type.—

Immediate acrid, metallic taste, with burning in gullet in a very few minutes (with arsenic the burning does not appear so soon);

Pain in stomach soon appears becoming intense in less than half-hour, and causing

Nausea and Vomiting, often with blood;

Purging, with tenesmus and blood-streaked stools, soon appears;

Collapse may ensue, but less likely to be profound than with arsenic;

(If a strong solution was taken, the mucous membranes of the mouth and throat will be whitened and shrivelled; with weak solution, inflammation and swelling including tongue and, sometimes, the glottis.)

Apparent recovery is likely to set in within a few days. This is the

2nd Period.—

The patient may feel quite well tho weak, and experience polyuria.

After 48 hours, or usually less, disturbances reappear constituting the

3rd Period.—

Oliguria or anuria set in, accompanied by development of

Entero-colitis,

Stomatitis, glossitis, laryngitis, and even edema glottidis.

Uremia, accompanied occasionally by nervous symptoms and cramps, then

Collapse or Coma, according to the intensity of the toxemia.

Death may follow in from 5 to 12 days of the time of taking the drug, or gradual recovery may be made.

Treatment.—

Egg white mixed with a little water, given at the earliest possible moment.

Promptly evacuate stomach either by emesis or by stomach tube.

Perform lavage, using albumen water. (Use great care if there is corrosion.)

Or, give large draught of warm (not hot) milk containing two teaspoonfuls of mustard powder. Follow with lavage if possible.

(The albuminous substance with which the mercury is precipitated **must not be left in the stomach**, as the mercury albuminate is capable of redissolving and being absorbed.)

Or, use "Mercury Antidote" (containing sodium phosphite and a citrate) put up in tablet form by Abbott Alkaloidal Co.

Continue demulcent treatment.

Control pain.

Manage collapse.

Anticipate uremia, and closely watch kidney condition, adapting treatment to findings, particularly during stage of euphoria.

The colitis will need special care, as will the stomatitis.

Tracheotomy or Intubation is occasionally needed.

Maintenance of nutrition during the long course of the condition needs particular care.

Sequelae.—Nephritis, entero-colitis, loss of teeth, neurasthenia.

Remark.—Note similarity to arsenic poisoning. Arsenic, however, is more intense and more definitely affects the nervous tissues, altho the gastro-intestinal symptoms of arsenic are a trifle slower in appearing.

Mushrooms.

Different varieties produce differing symptoms. **Some contain an active alkaloid, such as muscarine**, similar to but much more toxic than pilocarpine. **Others contain a substance having the characteristics of the bacterial toxins, and requiring an incubation period of from 12 to 18 hours for its effects to appear.** Both types tend to produce violent cramps, diarrhea and vomiting. Those of the muscarine type cause bathing of the body in perspiration, due to stimulation of gland nerve endings; and slow or irregular heart action, due to poisoning of vagus endings, after period of excitation, consciousness remaining clear. Those of the toxic, and **more common type**, add to the gastro-intestinal irritation symptoms, cerebral excitement to the extent of delirium, oliguria or anuria, hemolysis giving rise to hemoglobinuria and a subsequent jaundice, and poisoning of the heart muscle. Both, if serious, terminate in **collapse**, but the muscarine type would display contraction of the pupil, the latter a probable (tho not certain) dilatation of the pupil.

Treatment.—**Muscarine type:** physiological antidote, atropine used with caution, following the usual emptying of the stomach and bowels, and the symptomatic treatment for pain, and collapse.

Toxin type: empty stomach and bowels, control pain, delirium, and collapse. No specific treatment.

Nitric Acid, see "ACIDS."

OXALIC ACID.

Source.—Often found about households, where it is kept for use in cleaning woodwork, straw hats, etc., and for the removal of ink stains on cloth. Its **resemblance to Epsom Salt** has resulted in its having been taken accidentally in a number of cases. Ease of securing and knowledge of poisonousness lead to its being occasionally taken with suicidal intent. **A moderately frequent poison.**

Fatal Dose.—One-half to one ounce in medium strength solution is the most fatal quantity. Larger quantities are more likely to result in effective vomiting. Mortality probably about 35% unless treatment prompt.

Action.—**Locally**, corrosive if taken in crystalline form or as strong solution; so death may result in shock within 15 minutes.

Systemically produces **acidosis** and removes soluble calcium salts from tissues resulting in successive symptoms of partial then complete **calcium starvation**, according to amount absorbed. When death results from this cause, usually inside of 24 hours, and due to depression of nerve centers and heart muscle, with suppression of urine, patient being in coma at the last.

Symptoms.—**Immediate intense sour taste**, sense of heat in **stomach** within a few minutes, changing in a short time to a **burning pain**.

Mucous membranes of mouth and pharynx red and swollen, changing within an hour or so to a dirty gray in patches. This local injury causes **sense of constriction in throat** and increasing huskiness of voice, which increase due to systemic action upon pharyngeal muscles.

Vomiting within 10 or 20 minutes; apt to be severe and persisting. Vomitus of "**coffee-ground type**," separates on standing into a lower layer of altered blood and an upper lighter-colored liquid.

Shock and Collapse, followed by death may occur early or late. Instead of shock and collapse, sometimes without vomiting, when taken diluted there may develop within an hour or more, the **systemic action**;

Muscular twitchings, numbness;

Muscular cramps with pain, in extremities and lumbar muscles;

Muscular rigidity; blueness of finger-nails, lips and cheeks, from interference with respiration and circulation;

Convulsions. Finally in severe cases.

Suppression of urine from parenchymatous nephritis, or circulatory changes.

Coma.

Immediate Treatment.—

Give 2 or 3 heaping tablespoonfuls of **powdered chalk mixed to a cream with water** and then stirred into a tumbler of tepid water.

(Or, 1 or 2 teaspoonfuls of **Calcium chlorid** or lactate dissolved in a glass of water.) (Or, 1 heaping tablespoonful of Epsom Salt, Magnesium Sulphate.)

If these antidotes are present and are promptly used it is not necessary to wash out the stomach, altho this may be done with advantage if there is no sign of corrosion.

If **systemic symptoms** commence to appear, **administer slowly intravenously**, 500 cc. of water containing 2 gm. sodium chlorid, 5 gm. of sodium bicarbonate and 1 gm. of calcium chlorid.

Treat collapse according to indications.

Until stomach has been emptied, avoid use of **sodium or potassium compounds** or large quantities of **water**, as these increase absorption of oxalic acid.

If irritant action has not been severe, and if it is likely that some of the drug has entered the intestine, give full dose Castor Oil.

After Treatment and Sequelae.—

Endeavor to **avoid kidney complications** by use of **diaphoretic measures** and an abundance of water with small doses of sodium bicarbonate.

Several day's to a month's care will likely be needed to secure recovery from renal and gastro-intestinal irritation. Severe strictures of gastro-intestinal tract as after mineral acid poisoning, are rare in this case. The general care, however, is the same.

Tetany and acidosis may need treatment.

Remarks.—Dilute solutions of oxalic acid may give rise to symptoms entirely unlike those of the gastro-intestinal irritants, resembling rather during the early stages the nervous excitants, and during the later stages the vital depressants.

Phenol, see Carbolic Acid.

PHOSPHORUS, (Yellow Phosphorus).

Source.—Almost always matches or “rat poison.” “Rat poison,” which usually contains about 3%, is the more dangerous because of the emulsification of the drug and the absence of oxidizing chemicals as in matches. Matches are becoming a less common source, as the newer varieties contain non-poisonous red phosphorus, or some compound, and safety matches contain none.

Sometimes taken to produce abortion. Formerly quite common for suicidal purposes.

Fatal Dose.—Adult, 1 grain or more ($\frac{1}{2}$ to 1 teaspoon of rat poison).

Child, as little as $\frac{1}{5}$ grain. Adult, 100 or more old type matches. Mortality about 70%.

Action.—Usually slow. Internally it produces no effect like the external burning. Is **non-corrosive** and has no action until absorbed into the tissues. Death in less than 12 hours rare, and then due apparently to cardiac paralysis. Essential action is interference with internal oxidations of cells, resulting in the degeneration of those which are reached by any more than the merest traces of the drug. This explains the pathological conditions set up and the symptoms developed. Death usually due to **severe metabolic disturbances or degenerations of vital structures**, or of blood vessels, after disappearance of primary period of gastro-intestinal suffering. Most common time of death between second and seventh day.

Symptoms.—**First Period.** May be taste of garlic at time of ingestion, followed by odor of garlic on breath. Both of these may be lacking.

Abdominal pain and

Vomiting, after a period of from 2 to 6 hours after ingestion. (These are due to non-inflammatory changes in the gastro-intestinal mucosa, as the drug acts upon the cellular elements as it is absorbed into them. Membrane is pale rather than red, but shows areas of extravasation of blood due to degeneration of blood-vessel walls; and, if action is extensive, there will likely be bleeding into stomach and intestines. As absorption is completed or the poison is purged away, this primary period of action subsides.)

Vomit possesses **garlicky odor** and is **luminous** in the dark, unless considerable time has elapsed. If dose was in readily absorbable form, vomit is likely to contain **darkened blood**.

Purging a few hours later, dark in color (altered blood) and offensive.

During this period also there is likely **frontal headache**, **subnormal temperature**, and other symptoms resembling **mild degree of collapse**.

In less than three days, usually, patient appears to be recovering, when second stage appears. Sometimes first stage is so mild that no physician is consulted, and he first witnesses second stage, 36 to 72 hours after the dose was taken.

Second Period.—**Jaundice**, **hepatic tenderness**, increase in size of liver.

Often **recurrence of vomiting and purging**, with altered blood.

Urine shows reduced quantity of urea, marked increase of amino acids and ammonia compounds, showing increased protein destruction with incomplete oxidation; sometimes bile-tinted.

Marked tendency to **hemorrhages** from various mucous membranes, and even into the various organs; abortion of pregnant women.

Symptoms of greatest variety because of fatty degeneration in widespread organs of body, including the nervous system, with tendency toward tissue hemorrhages. **Acidosis** and symptoms of acute yellow atrophy of liver common.

Death by hemorrhage, collapse, coma, cerebral hemorrhage, or other cause.

Or, gradual recovery during second to fifth week.

Immediate Treatment.—If there is history of taking poison, do not wait for symptoms.

Phosphorus tends to remain in the gastro-intestinal tract for many hours, therefore:

If stomach is full of food administer as emetic and chemical antidote, 3 grains of **Copper Sulphate** dissolved in half glass of water, repeated every five minutes until free vomiting. (Forms insoluble copper phosphide.) Then

Wash out stomach with warm water until return water is clear. Then

Wash out stomach with about 8 gallons of 1:2000 solution of **potassium permanganate** (one-half dram to the gallon) to oxidize poison, which may be attached to stomach wall. With water wash stomach clean.

Give cathartic. Best, if it can be secured, **Old Oil of Turpentine** (an oxidized oil, which still further antidotes phosphorus), one-half dram mixed with one ounce mucilage of acacia, every half hour till four doses. **AVOID ALL OTHER OILS**, as they increase absorption of phosphorus. If old oil of turpentine is not available use Epsom Salt as purge.

Flush out colon. If odor or luminosity of phosphorus present, wash out colon several times with 1:2000 potassium permanganate solution.

Manage collapse, if present, as usual. Excessive pain, also.

After Treatment.—**Manage acidosis.** Check acidity of urine quantitatively and keep degree of acidity low.

Careful nursing and confinement to bed for several days at least.

Bleeding, then **transfusion of blood** sometimes valuable because of destructive action of phosphorus on patient's blood during early hours of poisoning, and frequent loss of blood during second period.

Mortalities are common after apparent beginning of recovery. Therefore, most careful study of patient's condition is required, and the prompt recognition and meeting of the needs of the body in the manifold complications.

Sequelae.—Disseminated forms of neuritis or paralysis occasionally.

Remarks.—Phosphorus is not a true gastro-intestinal irritant, but its symptoms most closely resemble those of this group. It may be classed as **one of the specific irritants**. In a case with mild initial symptoms it may be impossible to differentiate from acute yellow atrophy of the liver, unless phosphorus be found in the vomitus or dejecta.

Potassium Chlorate.

This substance has been the cause of a considerable number of deaths, as a result of its having been **swallowed when in use as a gargle**, or as a result of prescription of an overdose for internal administration. The probable fatal dose is 3 drams for an adult, 1 dram for a small child.

The strong solutions act as a direct **irritant to the gastro-intestinal mucous membranes**, causing the typical symptoms of this group. **Following absorption methemoglobinemia and oligocythemia** develop during the course of a few days. The same fixative action exerted upon the oxygen of the hemoglobin is probably exerted upon the peroxidase substances that appear essential to cell metabolism, with a resultant widespread injury to the functioning cells of the body. Disturbances of the liver, kidney, heart, spleen, and nervous system have been distinctly recognized.

There is **oliguria, hematuria, finally anuria, and**

Uremia. This added to the primary irritation of the nerve tissues causes **muscular spasms, delirium and coma**.

Collapse may have taken place earlier from cardiac poisoning, or in rare cases from the exhaustion of the gastro-intestinal irritation. The **skin may show varied discolorations, icteric or leaden**.

Treatment.—Empty stomach, by emetic or lavage. There is no antidote.

Give demulcents.

Manage collapse.

Use diaphoretic measures to assist elimination.

Anticipate and treat uremia.

Oxygen inhalations may assist the patient, but uncertain.

Transfusion of blood after slight bleeding may succeed in saving an otherwise hopeless case, by replacing the destroyed hemoglobin.

Potassium Hydroxid, see "ALKALIES."

RAT POISON, see Phosphorus, and Arsenic.

Sulphuric Acid, see "ACIDS."

Tartar Emetic, see Antimony.

Zinc Compounds.

Zinc Chlorid;

Soldering Fluid, ZnCl_2 with HCl ;

Zinc Sulphate, **White Vitriol**.

These substances are **relatively rare** causes of death. **The Chlorid is a true corrosive.** Symptoms of the strong mineral acids are duplicated by it, death being usually by shock or collapse. About the only points of difference are the styptic taste of the zinc salt, the lesser degree of corrosion, and the greater tendency toward recurrence of the symptoms after beginning recovery, due to the corrosive influence being carried further into the intestines. **Treatment as for Acids.**

The Sulphate is merely an irritant, therefore slower in producing the type symptoms, an hour or so being required. There is also added a set of symptoms from the **systemic action**: salivation, vertigo, cramps and convulsions.

Treatment.—Give egg white in water, or milk, as much as can be taken. If these materials are not vomited, they must be **removed by stomach pump**, as zinc albuminate is capable of being redissolved and absorbed to produce systemic symptoms. **Lavage** with water alkalized with **sodium bicarbonate**, or with strong tea will help to remove and render insoluble the remaining traces. After thoro cleansing of the stomach, milk and egg white freely may be given to be retained to serve as demulcents. **Control pain and collapse** or other symptoms.

GROUP II.

Central Nervous System Irritation.

Immediate symptoms.—Usually a selection of
Cerebral excitement, restlessness,
Abnormal muscular activities, convulsions,
Delirium,
Full pulse and hot skin until collapse ensues,
Coma.

With the volatile oils, added to the above are the
Gastro-intestinal and genito-urinary groups
of symptoms.

After symptoms.—Insomnia, nervous irritability.

General Treatment.—Empty stomach if poison taken by
mouth.

If an alkaloid, give tea or tannic acid solution as a
chemical antidote.

Physiological antidote in the form of an antagonistic
drug is usually prescribed but is often more in-
jurious than beneficial.

Use the methods cited under delirium, convulsions,
cardiac failure and other symptoms in the section
of the general management of poisoning.

ATROPIN and its Relatives

Hyoscyamin,
Hyoscin,
Homatropin.

Atropa Belladonna (deadly night-shade),
Scopola (Japanese belladonna),
Hyoacyamus niger (henbane),
Datura Stramonium (thorn-appale),
Duboisia,
Mandragora officinalis (mandrake).

Source.—Poisoning rarely by one of the atropic plants. Us-
ually from **excessive medicinal dose** (suppositories,
patent asthma cures, mydriatic use in ophthalmic
work).

Fatal Dose.—By mouth 1 grain, by hypodermic 1/10 grain of
the alkaloid likely to produce death; but there
have been many recoveries from these doses.
Mortality in only 10% of cases where symptoms
develop.

Action.—Many of the symptoms are due to the initial vagus ending paralysis followed by sympathetic paralysis, but the cause of death and of the dangerous symptoms is usually the central depression after exhaustion from the excitement stage. In some cases where the heart action is continued excessive for a long period cardiac failure may be the cause of death, but more frequently respiratory failure is the immediate cause. If death, usually in less than 24 hours.

Symptoms.—Cerebral:

Giddiness, distress from dry throat and inability to swallow.

Diplopia and other visual disturbances,

Speech wandering,

Fantastic hallucinations,

Violent delirium (rarely muttering), gradually changing to

Stupor or Coma, according to seriousness of condition.

Objective:

Dry mucous membranes,

Spasmodic movements of pharynx when attempting to swallow,

Eyes staring,

Pupils widely dilated, until sympathetic paralysis also effected,

Blood-pressure high, pulse very rapid,

Face displays scarlatinal rash, veins of forehead distended,

Skin hot and dry,

Body temperature elevated until collapse or coma set in, when

Pulse becomes irregular, slower and weak,

Respiration depressed.

Treatment.—Wash out stomach with tannin solution, 1 dram to a pint of water, in case drug was given by mouth. Emetics usually fail to operate. If dose hypodermically, emptying the stomach of little value.

Physiological antidote, formerly taught to be morphine, but this drug is now recognized to be contraindicated because it tends to hasten coma and to increase depression of the respiratory center. Chloral and chloroform contraindicated because of heart depression.

Delirium may be reduced by ether inhalations. Use ice cap, cool sponging to reduce body temperature, or more vigorous antipyretic measures as the evaporating wet sheet pack or the cold rubbing bath, using mild degrees of coolness. Properly adapted to the patient, these would be also sedative.

Heart condition can be improved by ice-bag to precordia during stage of vagus paralysis, for the purpose of depressing the accelerator nerves, and diminishing the tendency to cardiac exhaustion.

Chipped ice may relieve suffering from mouth and throat dryness.

Manage respiratory depression and collapse as they appear.

Barium Salts.

May resemble the action of a poison of this group when the dose is too dilute to produce gastro-intestinal irritant symptoms, because of its **high blood-pressure and hallucination** production. A dose of Glauber's Salts to clean out the colon, would incidentally antidote any barium compound in the alimentary tract.

Caffein.

This drug probably could scarcely cause poisoning in an individual with a normal heart. A very large dose, say of a dram, might cause such overaction of an already degenerated heart to a degree that might result in cardiac failure.

Symptoms would be mental confusion, restlessness, muscular tremors, possibly convulsions, and collapse.

Treatment.—that of collapse. The administration of an abundance of water containing a little salt, or saline irrigation of colon (water, 1 gallon; sodium chlorid, 1 tablespoon; sodium bicarbonate, $\frac{1}{3}$ teaspoonful) will assist the elimination of the drug.

The stomach, of course, to be emptied, preferably by lavage, if likelihood of any drug remaining there.

Patient apt to suffer from insomnia and nervous irritability for many months.

Camphor.

Used as an abortifacient, taken suicidally and accidentally, camphor has been responsible for a small number of deaths. The fatal dose and the mortality rate cannot be stated.

Symptoms.—Pain in stomach if taken in solution. Cerebral excitement, characterized by incoherent talking, and restlessness, appears in a few minutes because of rapid absorption. Face flushed, pupils dilated, pulse full. Convulsions develop if dose large and death usually takes place in convulsions, tho coma may first develop. Camphor odor will be detected on the breath.

Treatment.—Empty the stomach. For control of cerebral excitement use hydrotherapeutic measures in preference to the bromids or chloral. Collapse may develop and need treatment.

Cantharides, or Spanish Fly.

Most cases follow an overdose administered for its aphrodisiac or its abortifacient effects. It is not a common form of poisoning. The quantity likely to produce death is about $\frac{1}{2}$ ounce.

Symptoms.—The combined symptoms of the severe gastrointestinal irritants and the central nervous system irritants.

Immediate burning pain in the mouth, throat and stomach; vomiting, purging (often with stools bloody), tenesmus and collapse with shock.

Superimposed on these are vertigo, delirium, convulsions, tending toward comatose exhaustion.

Genito-urinary symptoms are also always present: strangury, priapism, hematuria, suppression of urine, abortion of pregnant women.

Treatment.—Empty stomach. Give mild saline purgative, with an abundance of water. Addition of a third of a teaspoon of salt to each pint of water drank may assist elimination.

Oils must not be given as they increase absorption.

Flush out the colon to remove any possible accumulation of poison there.

Give freely of non-oily demulcents.

Manage the symptoms as they appear, particularly the collapse and pain.

Anticipate and treat suppression of urine.

COCAINE.

Eucaïn,
Novocain.

Source.—Usually following injection of excessive medicinal dose. Relatively common.

Fatal Dose.—10 to 20 grains. Mortality even where poisonous symptoms develop rather low. Numerous instances are found where a small overdose gives rise to moderate symptoms from which patient readily recovers.

Action.—**Sudden death** within five minutes of the time of administration **sometimes** takes place in a condition of collapse or shock whose origin is unknown. **If death does not occur early in the poisoning, recovery usually follows during the succeeding 24 hours.** Occasional late deaths result from cardiac failure. Symptoms during the course of the poisoning are central, due to an atropin-like action upon the cerebrum and medulla, and peripheral, due to the action of cocaine upon the sensory nerve endings. Action upon the eye is rather stimulation of the sympathetic mechanism than paralysis of the oculo-motor nerve. Excessive quantities finally paralyze voluntary motor nerve endings.

Symptoms.—Giddiness, muscular incoordination,
Hallucinations and delusions.
Great anxiety and fear of death, which probably contribute to
Shock-like condition in which death may occasionally take place.
Throat dry, scratching sensation in pharynx.
Sensation as of worms beneath the skin (Magan's sign.)

Objective.—

Forehead covered with perspiration, shivering, skin usually damp and cold (distinction from atropin),
Pupils dilated but react to light,
Respiration quick and panting,
Pulse rapid with blood-pressure slightly raised,
Nausea and vomiting sometimes from central action,
Stage of excitement followed by stupor with occasional convulsions and
Coma, in extreme cases.

Treatment.—Empty the stomach, if the poison was taken by mouth.

Firmly reassure the patient of his recovery to offset the intense anxiety usual in this condition.

Promptly give the treatment outlined under **Cardiac Failure**, omitting the use of morphine.

Osteopathic manipulations thruout the upper dorsal and lower cervical regions have been seen by the writer to promptly check the prostration in novocaine poisoning.

Collapse treatment may be needed in the later stages, particular attention being given to the maintenance of respiration.

Sequelae.—Incompacitation for work for days or weeks.

HYOSCIN, see Atropin.

Iodoform.

Poisoning from this substance is almost invariably due to absorption from cavities which have been packed with iodoform gauze. Whenever, while this form of packing is in use, there develop headache, wakefulness, giddiness, delusions, delirium, suspicion should be aroused that poisoning is taking place. Occasionally the symptoms developed take the form of stupor instead of cerebral irritation.

Mushrooms.

Those varieties of mushrooms containing toxin-like substances may set up poisoning with the symptoms ushered in by delirium and convulsions, thus resembling members of this group. See **Mushrooms, Group I.**

Nux Vomica, see Strychnine.

Oil of Pennyroyal,

Oil of Tansy,

Oil of Turpentine,

Oil of Wintergreen, see **Volatile Oils,**

Salicylic Acid.

Poisoning almost always due to medicinal over-dosage or more rarely to use of excessive quantities as a food preservative. Occasionally the symptoms set up include restlessness, mild delirium, or mental excitation, which require its mention in this group.

Stramomium, see Atropin.

STRYCHNINE.

Nux Vomica,
Rat's bane,
Brucine.

Source.—About half of the cases are suicidal, one of the pharmaceutical preparations having been ingested. There are also numerous homicidal cases on record, less than one-third of all being accidental, most of these being due to errors in medication.

Fatal Dose.—Probably 1 grain, rapidly absorbed, would be fatal. Under certain conditions, recovery has followed the ingestion of as much as 20 grains, even though the drug remained in the stomach for a period of more than an hour before vomiting took place. Mortality 50% in case drug is absorbed and symptoms appear.

Action.—Due almost entirely to the extreme increase of reflex irritability of the spinal cord and, to a lesser degree, of the medulla and cerebrum, followed by paralysis or exhaustion of the nerve centers. Also some degree of paralysis of motor nerve endings. **Death due to spasmodic fixation of the respiratory muscles, or delayed paralysis, or exhaustion of the cord and medullary centers; or, if duration of the case is prolonged, from cardiac exhaustion.** Recovery usually follows if patient lives for five hours after onset of symptoms. Fatal cases most frequently terminate during the fourth or fifth tetanic seizure of the attack.

Symptoms.—In a typical case all other symptoms are masked by the muscular convulsions which develop. There may be, at the outset,

Muscular twitchings of various groups of muscles followed soon by

Tetanic Convulsions.

Opisthotonus, a condition of extreme, persisting contraction of practically all the extensor muscles of the body,—head thrown sharply back, body arched backward, soles of the feet arched inward, etc.

Risus Sardonius, or hideous distortion of the face, with the angles of the mouth retracted,

Trismus, and numerous other less characteristic muscular distortions are produced. During the spasm, the thoracic muscles, diaphragm, and abdominal muscles are rigidly set, thus stopping respiratory movements.

Relaxation usually takes place in a few minutes. Soon however, in response to some peripheral stimulus (the jarring of a door, sudden noise, a draft of air) another spasm is produced. From three to ten such spasms take place in an ordinary case. The duration of the spasms may be from a half a minute to fifteen minutes and the interval between them from one minute to an hour. In cases likely to terminate in recovery, the interval between the spasms is lengthened and the intensity of the spasms diminished.

Consciousness usually remains clear excepting upon the approach of exhaustion or death. Between the spasms there is profound muscular relaxation due to exhaustion.

Treatment.—If it is discovered that strychnine has been taken by mistake before symptoms appear, the throat should be cocainized, the stomach tube introduced, and the stomach washed out with a solution of tannic acid, 1 dram to a pint of water. Lavage should be continued until the returning fluid is clear. During the latter part of the procedure potassium permanganate (6 grains to the pint) solution should be used, if possible, in place of tannic acid solution. A mouth gag must be used to prevent possible biting of tube in two.

After symptoms have developed, use of the stomach pump can be managed only after anaesthetizing the patient.

The convulsions may be controlled by ether or chloroform administration if the patient is able to anticipate the arrival of a new spasm, as is sometimes the case. The continuous use of the general anaesthetics increases likelihood of paralysis of the respiratory center.

Sodium Bromide, 1½ drams in a solution by mouth or by rectum is usually advised. **Spinal anaesthesia** has been suggested but not successfully used. **Morphine** is contra-indicated.

Artificial respiration, smoothly and rhythmically given, between convulsions, seems to diminish their frequency, probably by preventing increased irritability of the nerve tissues due to carbon dioxide.

Intravenous infusion of Ringer's solution seems to accelerate the elimination of the drug. **Catherization** may be necessary to prevent resorption from the bladder. **Intratracheal insufflation** has also been recommended.

It is suggested that during a prolonged spasm, wrapping the patient in a hot blanket pack might shorten its duration.

Avoid startling patient, inform him of every move that is to be made and protect him from all noises, jars, and sudden movement. These measures seem to be of great importance in determining the severity and frequency of the spasms.

Sequelae.—Exhaustion of the heart muscle requiring prolonged rest; prolonged nervous irritability.

Turpentine, see Volatile Oils.

VOLATILE OILS.

Oil of Turpentine,
Oil of Pennyroyal,
Oil of Wintergreen,
Oil of Tansy, etc.

Because of their reputation as **abortifacients**, these drugs are sometimes responsible for death. Occasionally **over-dosage**, when used as an athelminic is responsible for poisoning. The symptoms and treatment of poisonings by any of these volatile oils will be the same.

Symptoms.—Burning pain in mouth and throat,

Vomiting,

Diarrhea,

Difficult mictuition, strangury with hematuria or albuminuria,

Mental excitement,

Muscular spasms,

Coma or Collapse.

Each of the volatile oils possesses its own characteristic odor and tends to produce its characteristic odor in the urine.

Treatment.—Empty the stomach.

Give saline purgative if diarrhea has not already occurred.

Give demulcents,

Manage collapse,

Anticipate and treat suppression of urine.

Sequelae.—Menorrhagia, cystitis, nephritis, entero-colitis, and nervous irritability.

GROUP III.

Vital Depression.

The members of this group are arranged according to the following outline, and are placed in the order in which they conform to the type:

Physical weakness not due to shock,—

(1) with drowsiness or stupor from cerebral depression

Morphin and Opium preparations,
Chloroform,
Chloral,
Ethyl Alcohol,
Methyl Alcohol,
Sulphonal, Veronal, etc.,
Acetanilid, Antipyrin, Phenacetin,
Dilute Phenol, Lysol, Cresol.

(2) with consciousness retained

(a) weakness due to motor nerve paralysis

Conium, or hemlock;
Curare, or arrow poison;

(b) weakness due to spinal or medullary depression with deficiency of circulatory and respiratory functions

Physostigmin, or Eserin;
Muscarin, or mushroom;
Nicotin, or tobacco;
Aconite, or monkshood;
Veratrum viride;
Digitalis;
Ergot;
Gelsemium.

(The latter members of this group display some cerebral depression; in poisoning by the earlier ones of the list unconsciousness occurs sometimes from syncope.)

(3) preceded by epileptiform convulsions and due to tissue asphyxiation.

Hydrocyanic acid, and the Cyanids,
Nitrobenzene.

Treatment of these forms of poisoning is largely symptomatic. The antidotes most frequently of value are tannin (1 dram to the pint of water); or potassium permanganate (about 20 grains to the gallon), used in lavage. It is best to leave neither of these in the stomach.

Collapse, Cardiac Failure and Respiratory Failure treatments will be very frequently needed.

The physiological antidotes as a rule merely complicate the condition; because they are given without considering any but the immediate action, both facts being ignored that the poison will have shortly changed the patient's condition (a vagus action disappearing, for example, and being replaced by exhaustion of center) and that the antidote will have a remote effect as well as an immediate effect. Use guarded physical methods instead of drugs.

The sequelae are varied according to the various poisons.

SUB-GROUP (1).

MORPHIN.

**Codeine,
Heroin,
Laudanum, and other
Opium Derivatives.**

Source.—Poisoning is seldom homicidal, frequently suicidal, sometimes accidental, particularly from overdose to children. An occasional death is due to the excessive indulgence among habitues.

Fatal Dose.—For an adult 3 grains is about the smallest possible fatal dose, as much as 60 grains having been recovered from. Habitues frequently take without danger upwards of 30 grains. In infants so small a dose as $1/12$ grain has frequently caused death. Death usually occurs between the 12th and 24th hours in fatal cases, rarely in less than one hour.

Action.—There is initial, transient excitation of the central nervous system, followed by depression of the cerebrum and medulla, particularly involving the respiratory center. There is also a peripheral effect checking peristalsis, after a period of increase, and resulting in marked intestinal stasis. Some interference with the action of the micturition center prevents the evacuation of the bladder which, unless artificially emptied, may even rupture. Death is from asphyxia due to paralysis of respiratory center.

Symptoms.—Within about one-half hour of time of taking dose by mouth

Excitement stage developes,—characterized by physical activity, talkativeness, **vivid imaginativeness**, hallucinations. In habitues this stage is more marked; in children spinal excitation may be shown by the appearance of convulsions; in ordinary adults the excitement usually passes off in a few minutes, altho there are rare cases of almost maniacal excitement. The

Stupor period begins with a sense of weariness and irresistible drowsiness, if permitted, the patient falling into a profound tho apparently normal type of sleep. But the face becomes pale, skin bathed in cold perspiration, pupils contracted, and often an erythema develops. If opium were used its odor could be detected on breath.

Narcosis, from which the patient cannot be aroused, gradually develops. At this period there is usually

Profound muscular relaxation and dropping of the jaw;

Leaden colored face, and blue lips; pupils.. “pin-point” in size;

Respiration grows slower and more shallow, stertorous or of Cheyne-Stokes type;

The pulse slows and weakens until imperceptible.

As death approaches there may be a few transient convulsions of probable asphyxial origin, preceding death.

Cessation of respiratory movements occurs before heart stops beating.

Treatment.—Lavage with potassium permanganate solution, 20 grains to gallon. This is needed whether drug was administered by mouth or by hypodermic; for excretion of the drug into the stomach takes place very actively, and if it be not removed it will be absorbed from the intestines later.

Flush out the intestines also to assist in elimination.

If patient found in the stupor stage, awaken if possible and keep him walking about between a pair of attendants, to prevent lapsing into coma. This procedure is important, perhaps the activity affecting the elimination from the system; but if patient is incapable of co-operation, it is probably better to not force upon him the exhaustion which may be developed from forced activity.

Stimulant treatment of varied forms is recommended.

Probably more rational and effective than most given are those given in this outline under treatment for shock, page 37. Flagellation with wet, cold towels is often mentioned in the older texts. Of the numerous drugs, atropin seems worse than useless. Caffein comes the nearest to supplying a stimulation of a desirable nature.

Artificial Respiration is very important. The faradic method is well suited in this condition. Oxygen may be used with advantage.

Intubation may be necessary because of relaxation of throat muscles.

Venesection followed by compound saline infusion aids elimination.

Catheterization of bladder to prevent distention should be remembered.

Treatment should be long continued, for life often reappears where it seems certain to have ceased. There being no injury to the heart in most cases, it will continue work so long as it is supplied with oxygenated blood. Artificial heat, and slight elevation of the limbs may assist to a continuance of a weakening heart action.

Chloroform.

Source.—Apart from deaths during surgical anesthesia, most of the deaths from inhalation of chloroform are among habitues or those who use it frequently for relief from spasmodic affection of some sort. Deliberate suicidal attempts with chloroform are made by drinking the drug. Of the attempts usually about 30% are successful.

Fatal Dose.—The quantity necessary to cause death by inhalation cannot be stated, as it depends rather upon the rate of absorption than upon the total quantity. By mouth 1 ounce may be fatal, tho recoveries have often followed larger doses.

Action.—The action in surgical anesthesia can not be more than mentioned here. In this case death is usually within the first few minutes and is due to combined reflex medullary stimulation and direct toxic action upon heart muscle by drug in freshly saturated blood from the pulmonary bed. Excepting for the reflex effects and the greater in-

fluence upon the heart when the drug is inhaled, the action is the same as when introduced into the blood stream thru the stomach. There is general central nervous system depression, with particular effect upon the respiratory center. Degenerations of the liver are more apt to follow administration by stomach. Death in this case in 12 to 36 hours.

Symptoms.—When poison taken by mouth, odor usually evident on breath.

Burning sensation while swallowing, the irritant effect perhaps causing some reddening and swelling of mucous membranes.

Feeling of strength may be felt for a few moments. Then

Irresistible drowsiness and

Sudden development of unconsciousness.

Respiration becomes slow and weak,

Skin cyanosed and covered with cold perspiration,

Body temperature lowered. Death takes place from

Cardiac failure combined with respiratory center paralysis, or recovery may follow, gastro-intestinal irritation becoming evident as the patient regains consciousness.

Treatment.—Empty stomach, preferably by lavage.

Stimulant treatment is usually prescribed,—whiskey, strychnin, strong coffee enemas, etc. It is recommended that the regular treatment for collapse be followed here, remembering the danger of adrenalin and of too much lowering of the head in this condition. Oxygen inhalations and artificial respiration are of fundamental importance in many cases.

Chloral Hydrate.

Source.—Most cases are accidental poisonings among habitues. Some are due to over-dosage medicinally, some to suicidal effort, some to criminal intent to stupefy the intended victim of some attack. There are more chloral than chloroform poisonings.

Fatal Dose.—Great variability. Death has followed a medicinal dose of 20 grains and recovery has followed dose of 720 grains. Among attempted suicides there is a mortality of only about 20%.

Action.—Appears to be identical with chloroform after absorption, but absorption much slower. The irritant effect locally is less, the central effect more gradually developed, and the toxic action upon the heart not quite so marked. Death from cardiac failure or pulmonary edema from prolonged defective heart action and respiration. Time of death usually 12 hours, may be 2 to 48.

Symptoms.—The same as those for chloroform, taken by mouth.

Odor of chloral may be detected on breath of patient.

Important to distinguish from morphin, because in morphin, it is safe to arouse and exercise the patient, while in chloral stupor such a procedure might precipitate heart failure. Pupils of eyes in chloral poisoning not pin-point, and respiration not so profoundly slowed as with morphin.

Stupor may resemble normal sleep, but for the slower respiration, and may be spontaneously awakened from after as long as three days.

Treatment.—Empty stomach.

Maintain body warmth and respiration.

General collapse management.

ALCOHOL.

Grain Alcohol, or Ethyl Alcohol.

Whisky, Brandy, Wines, Etc.

Source.—Occasionally are found cases dependent upon the accidental ingestion of excessive quantities, followed by poisoning, usually among children. Usually the cases met are those of excessive, intentional indulgence in the beverages. Occasional suicidal cases are met. Causes a large number of deaths yearly.

Fatal Dose.—Of pure alcohol about 6 ounces for an adult, 3 ounces for a child. Of the beverages, that amount which would contain about the preceding amounts, if taken within a short period of time.

Action.—Alcohol is locally irritating, depresses the entire nervous system, in large doses depresses the heart muscle and the muscle of the arterial walls. Prolonged use results in excessive irritability of the nervous system, rendering possible such conditions as delirium tremens, or an acute mania.

Symptoms.—The intoxication stage, present while only the higher areas of the brain are depressed, and characterized by the familiar symptoms of muscular incoordination, mental incoordination and depressed sensibility, needs no description here. Following this the individual enters the stage of stupor. In this period there is muscular relaxation, bloated face, noisy breathing, flushed skin, full but compressible pulse. It is possible by vigorous efforts to rouse the patient, who may then be capable of making answers to questions, but the answers will be stupid and incoherent, in contradistinction to the intelligent responses secured from a morphin case when aroused. In the alcoholic the pupils will be dilated instead of constricted. If the quantity of alcohol ingested was greatly excessive the stupor may deepen to coma, which may pass into collapse, because of the weakening circulatory system. Death when it occurs is usually from collapse. In some cases the local irritant action is sufficient to induce shock.

Treatment.—Empty the stomach, preferably by stomach tube. It is better to avoid the use of apomorphine, because of its depressant effect upon the circulation.

Wash the stomach until no odor of alcohol is left in the washings.

Coffee in which is dissolved two teaspoonfuls of magnesium sulphate may be introduced into the stomach before withdrawing the tube.

If the patient is in a collapse condition, this should be treated as previously outlined.

Catheterization should be performed from time to time.

Delirium Tremens.—Is usually a symptom in chronic alcoholics when a drinking bout has been suddenly interrupted, or when while drunk an illness or an injury overtakes them. The condition needs no description, except to note that this condition is usually marked by a febrile temperature instead of a lowered temperature, as in ordinary alcohol poisoning. It is usually of sudden onset.

The treatment is largely symptomatic to control the insomnia, the physical excitement, and the mental disturbances. The former procedure has been

to unhesitatingly prescribe hypnotic drugs. It is now being recognized that this procedure caused an unnecessary mortality. The patient should be treated in an institution where there are proper facilities for hydrotherapeutic measures. Most important of these are the neutral bath and the neutral pack. Mechanical restraint must be avoided as it increases the death rate. If managing a case in a private residence the use of the hypnotics may be unavoidable; the bromids and a hyoscin containing preparation are the most frequently advised.

**Wood Alcohol.
Methyl Alcohol, or
Methylated Spirits,
Columbian Spirits.**

Source.—Usually due to its ingestion in adulterated whiskey or other substances, when grain alcohol in some form is desired. Some suicidal cases, and some accidental among children from drinking flavoring extracts or other preparations containing it.

Fatal Dose.—Probably 2 ounces would cause death in many cases.

Action.—The same as ethyl alcohol, but more highly toxic, more irritant and has an almost specific action upon the optic nerve. In the body formaldehyde is one of the oxidation products; this probably helps account for its greater systemic effect.

Symptoms.—Early nausea and vomiting much more marked than with ethyl alcohol. Then symptoms may be almost lacking for many hours or a few days. If the elapsed time is long, the symptoms are apt to be limited to the effects of nerve degenerations, particularly of the optic nerve. These include, color blindness, rapid loss of vision, which may be temporarily recovered and then again lost permanently.

When the symptoms of systemic action set in more early, they may progress rapidly to coma or collapse. Deaths are quite frequent.

Treatment.—Empty the stomach as early as possible and perform lavage. Pilocarpine chloride ($\frac{1}{4}$ grain) is most widely recommended as a diaphoretic to assist elimination. The depressant action of this drug upon the circulation should throw doubt upon the wisdom of its use. There is usually time in the management of this form of poisoning to utilize some of the more rational diaphoretic measures.

Collapse is likely to need careful attention. Proctoclysis of saline solution will assist both elimination and postponement of collapse.

**Sulphonal, Veronal,
Trional, Tetronal.**

Source.—Usually from excessive use to induce sleep.

Fatal Dose.—Depends upon patient's condition. Death has resulted from 30 grains, recovery has followed 30 times this amount.

Action.—Depression of cerebrum, gastro-intestinal irritation, and renal irritation produced.

Symptoms.—Stupor, with mental confusion and hallucinations if absorbed in large quantities.

Muscular weakness.

Nausea, vomiting and diarrhea, sometimes.

Suppression of urine and uremia, at times.

Collapse.

Treatment.—Empty and wash out stomach and bowels.

Guard condition of kidneys.

Manage collapse and other symptoms. Caffein and other stimulant drugs often recommended.

Acetanilid, or Antifebrin ;

Phenacetin ;

Antipyrin ;

Anilin, Pyridin ;

Dilute Carbolic Acid (Phenol),

Lysol, Cresol, Etc.

Source.—Usually from injudicious medicinal use. Rarely suicidal.

Fatal Dose.—From 20 to 30 grains up, the smaller doses having toxic action only occasionally.

Action.—Gastro-intestinal irritation, central nervous depression, renal irritation, cardiac depression following stimulation, and formation of methemoglobinemia by many of group.

Symptoms.—Variable. Most important are
Mental dulness and tendency toward
Collapse.

There also occur with some of the preparations, abdominal pain, vomiting and diarrhea, convulsions of cerebral origin, suppression of urine, erythema and cyanosis.

Treatment.—Empty stomach and bowels.

Manage collapse, respiratory and circulatory failure, and suppression of urine as they develop.

SUB-GROUP (2).

Conium, Poison Hemlock.

Source.—The infusion of this drug was used as a judicial poison among the Greeks, and its use in the execution of Socrates has given it an undying popularity as a classical topic for discussion in toxicology. It is now a very rare poison, a few deaths having occurred from it during the nineteenth century, from eating the plant root in mistake for parsnips, or otherwise accidentally ingesting it.

Fatal Dose.—Is about 3 minims of the alkaloid.

Action.—Essentially a motor nerve ending paralysis combined with medullary center depression, but with no cerebral depression. The respiratory failure from the motor paralysis will, of course, introduce in the patient's condition numerous distressing secondary symptoms.

Symptoms.—While consciousness is preserved until near the time of death, there is utter inability to move. On ingestion there is a burning or scratching sensation in the throat, soon followed by a sense of dizziness. The sensation of prickling in the extremities, accompanied, perhaps, by a few mild spasms, is soon followed by loss of muscular power, so that walking would be difficult, speech becomes indistinct, deglutition difficult. Finally, with loss of respiratory actions and medullary functioning, a complex set of symptoms develop which will be largely determined by the treatment being administered.

Treatment.—Most essentially the application of artificial modes of respiration. There is no physiological antidote. Removal of the contents of the stomach and bowels will have to be accomplished mechanically, by the stomach tube, because of loss of vagus and voluntary nerve functioning. Washing the stomach with tannic acid, 1 dram to the pint of water, or strong tea or coffee will precipitate any alkaloid that might be left in the stomach.

Curare, Arrow Poison.

Source.—This drug being used exclusively, in civilized countries, only in laboratory investigations in physiology, is an extremely rare cause of poisoning. It is much less toxic when taken by mouth than when introduced in the “natural” manner into a wound.

Fatal Dose.—Probably 3 grains of the alkaloid would be a fatal dose.

Action.—The primary action is a primary strychnin-like action upon the cord, followed rapidly by complete paralysis of the motor nerve endings in the voluntary muscles.

Symptoms.—After administration of the dose there is likely a short period of agitation and perhaps mild convulsions, followed by increasing relaxation of the muscular system, slowing of the respiration, and final death by asphyxia, which will excite numerous modifications in the patient’s condition from moment to moment. Consciousness remains clear until asphyxia develops.

Treatment.—Same as for conium. If poison was introduced thru a wound, ligation above the wound and encouragement of bleeding, as with snake-bite, would be indicated.

Physostigmin, Eserin, Calabar Bean.

Source.—Like the two preceding poisons, this is one which has long been known to the unscientific world, this one being in use in judicial proceedings among the natives of West Africa. Physostigmine, the alkaloid of Calabar Bean, is now a possible cause of accidental poisoning, because of its use in medicine.

Fatal Dose.—One grain would cause serious disturbances and possible death.

Action.—The action is stimulation of the endings of the vagus and the voluntary motor nerves, accompanied by depression and paralysis of the spinal and medullary centers. The heart is moderately depressed directly.

Symptoms.—As with the two preceding poisons, there is loss of muscular power, with consciousness retained, and death by respiratory paralysis. With physostigmin there is more resemblance of the condition of collapse, due to vagus stimulation and heart depression. Asthmatic breathing, abdominal cramps, vomiting (sometimes), pupilo-constriction, sweating, low blood-pressure with irregular heart are all largely due to stimulation of the endings of the visceral nerve fibres, and persist until the depression of the medulla sets in. Respiratory paralysis is the usual cause of death.

Treatment.—Should include attention to the collapse-like condition, the failing respiration and the cramps and broncho-constriction. In milder degrees of poisoning from excessive dosage of the drug, where vagus ending stimulation is most marked, atropin is the physiological antidote and will check the cramps and the asthmatic breathing, tho it increases the tendency to collapse in more severe cases. Artificial respiration should be persisted in when there is any chance of preventing asphyxiation.

(Conium, Curare and Physostigmine are all listed as paralyzants, but it is to be noted that the last does not paralyze nerve endings.)

Pilocarpin, Jaborandi.

Source.—Use in remedies for baldness renders it possible as a cause of accidental poisoning. Danger of a given dose varies much according to individual.

Fatal Dose.—Two grains would be fatal in many cases.

Action.—Resembles that of physostigmin, but more toxic to the heart, more stimulant to the secretory nerve endings, and less completely depressant to the spinal and medullary centers.

Symptoms.—Marked prostration from diminution of spinal tone combined with vagus slowing and direct depression of the heart. Heart slowing and weakening, combined with initial rise of blood-pressure from primary excitation of vaso-constrictor center, and interference with respiration due to broncho-constriction, bronchial secretion and loss of spinal tone, all tend toward edema of lungs, which is a prominent factor in causing death. There are also pupilo-constriction, profuse sweating, salivation, and possibly cramps, diarrhea and vomiting, all largely due to nerve-end stimulation. Consciousness is retained.

Treatment.—Atropin, in early cases where symptoms not too dangerous, to control cramps and asthmatic breathing. This “physiological antidote” will make the condition worse if depression of the centers is approaching.

Give treatment outlined for Cardiac Failure. Artificial respiration likely to be needed.

Mushrooms, Muscarine.

This alkaloid, not destroyed by cooking, occurs in certain varieties of mushrooms. The action is nearly identical with that given for pilocarpine. The treatment is the same.

Nicotine, or Tobacco; Lobelia, or Indian Tobacco.

Source.—Cases of nicotin poisoning other than the frequent mild poisoning resulting from the first efforts to acquire the tobacco habit, are of infrequent occurrence. They have occurred, however, from use of tobacco infusion as an enema, from use of tobacco as a poultice or to plug a wound, from the swallowing of the juice in tobacco chewing, from the use of “pipe oil” to induce abortion, and by a child’s sucking on an old tobacco pipe.

Fatal Dose.—The fatal dose of nicotine is about 3 grains; of tobacco, the amount varies according to its employment, 1 ounce of a strong infusion having caused death when used as an enema.

Action.—Depends upon paralysis of the sympathetic ganglia by depression of the pre-ganglionic synapse, accompanied by an initial cerebral and medullary stimulation of mild degree, followed by depression.

Symptoms.—Mild collapse with clear consciousness is the effect of moderate toxic doses. There are vomiting and diarrhea from medullary stimulation with early splanchnic nerve paralysis. The collapse induces tendency to recurrent syncope. Later and with larger doses there may be stupor from cerebral depression. Death in less than fifteen minutes may take place with large doses from profound syncope which is not adequately treated. Later death from apnea.

Treatment.—If possible, is first directed to remove the poison by methods according to the channel of administration. Manage the collapse, giving particular attention to maintenance of respiration and allowing a fair amount of stimulation by non-medicinal measures. Repose is essential to prevent syncope.

Aconite, Monkshood, or Wolf's Bane.

Source.—Poisoning usually due to accidental overdose or misuse for medicinal purposes.

Fatal Dose.—Of the alkaloid, 1/10 grain; of the crude drug, 1 ounce. Death may occur inside of one hour and is usual in fatal cases before five hours.

Action.—Is an initial stimulation followed by depression and paralysis of the medullary centers, accompanied by a direct toxic action upon the heart and a specific stimulation followed by depression of the sensory nerve endings.

Symptoms.—Tingling of mouth and throat, tingling of skin followed by chilliness, then anesthesia. The blood-pressure at first rises, there is asthmatic breathing from the broncho-constriction, and sweating, all due to the medullary action. With the depressent action there comes diplopia, tendency to syncope, respiration becoming slow and shallow, pulse becoming slow, weak, intermittent. Excepting for the periods of syncope, consciousness remains clear even during the collapse of the later stages. Death from asphyxia with cardiac failure.

Treatment.—Empty the stomach. Perform lavage with tannic acid solution (1 dram to pint) or solution of iodine, 12 grains, and potassium iodide, 30 grains, pint of water. Manage the collapse and the failing respiration. Atropine is recommended to control the asthmatic breathing and the slow heart of the early stages, but since the central action of atropine is about the same as aconite this would intensify the later symptoms.

Veratrum Viride,
Swamp Hellebore, or Indian Poke.

The action is somewhat different, the symptoms and management are the same as for Aconite.

There is collapse tending toward asphyxia with retained consciousness.

Digitalis, or Foxglove;
Digalen, Digitalin,
Digipuratum.
Strophanthus.
Scoparius, or Broom.
Scilla.

Source.—Excepting for the cumulative poisonings which these drugs give rise to, cases are rare.

Fatal Dose.—A small dose, not exceeding the average therapeutic dose, may cause serious symptoms when the drug has been in previous use. As a single dose a large amount is necessary to endanger life, probably $1/5$ grain of digitoxin, or 1 ounce of the tincture of digitalis.

Action.—Protoplasmic poisons. Gastro-intestinal irritation; medullary stimulation, depression, and paralysis; cardiac poisoning; renal irritation. Death by respiratory paralysis supplemented by severe and dangerous cardiac injury (affecting particularly the His-Tawara system). From a single dose poisonous action slow.

Symptoms.—About half an hour after taking dose a sense of fatigue and headache develop; nausea, vomiting and diarrhea may soon appear; vertigo, and if patient attempts to stand up, syncope. There is soon felt cardiac anxiety and a sense of suffoca-

tion of the case is long enough there may be altered. The pulse, at first slow, firm and regular, becomes irregular, intermittent, and weak, tho remaining slow. The respiration at first slow, and deep, becomes shallow and dyspneic. There is syncope upon the slightest effort. If the duration. Visual disturbances are commonly experibuminuria, or anuria. Altho there may be death in a few hours, it is more likely to be postponed for one to three days, when it takes place from heart failure.

Treatment.—If case seen early enough the stomach should be emptied and washed out. Atropin may control the early slow heart, but must be used cautiously because of its later medullary depression. Repose is most essential. The management of the collapse will have to be by the gentlest methods because of the heart injury, but warmth should be well maintained. Caffein is suggested for control of the heart block.

Ergot.

Source.—Poisoning usually from overdose taken as an abortifacient. The chronic form, ergotism, is almost unknown in this country.

Fatal Dose.—One-half ounce of fluid extract, possibly less.

Action.—Primary stimulation followed by paralysis of sympathetic nerve endings. Also a local protoplasmic irritant action.

Symptoms.—Nausea, vomiting, diarrhea; abortion; tingling of extremities; muscular cramps; collapse; sometimes in prolonged cases, coma instead of death in syncope.

This poisoning differs from the preceding by the mental depression which is lacking in the others.

Treatment.—Manage gastro-enteritis and collapse.

Gelsemium, or Yellow Jasmine.

Source.—Occasionally taken in mistake for some other medicine. More rare at present than formerly.

Fatal Dose.—Of the tincture 40 minims has caused death of an adult. It may be noted that this is only five times the U.S.P. dose.

Action.—From the standpoint of action, this drug should be placed near conium, which it resembles in its peripheral paralyzant effect; but its central depression causes stupor apart from tendency of the low blood-pressure and respiratory failure to cause unconsciousness. There is an appearance of the patient which closely resembles death while there is opportunity to avoid this issue. Death usually in five hours. 40% mortality.

Symptoms.—Giddiness, nausea, diplopia, swallowing and speech becoming impossible. Voluntary muscles less completely paralyzed than with conium, therefore possibility of convulsions due to early central action and tissue asphyxia. Stupor with imperceptible pulse and suspended respiration ensue. The patient may appear to have died, and yet recovery may take place spontaneously.

Treatment.—Empty the stomach. Withhold “physiological antidotes,” as all seem to make death more certain. Collapse treatment with moderate stimulation. Patient should be kept supine with head raised slightly, not thrown back.

SUB-GROUP (3).

Hydrocyanic Acid,

Prussic Acid,

Potassium Cyanid.

Oil of Bitter Almonds, Cherry Kernels.

Source.—Potassium Cyanid is used in the recovery of gold from ore, in photography, in gold and silver plating, and in certain manufacturing processes. The hydrocyanic acid gas is liberated in connection with electro-plating, in tanneries, in smelting processes and other places. But because of the care used in the use of these intense poisons, poisoning from them is rare, excepting when it is used deliberately with suicidal intent.

Fatal Dose.—Of pure hydrocyanic acid, 1 grain; of potassium cyanid, 2 to 20 grains; of oil of bitter almonds, 1 to 4 drams. The quantity required to kill by inhalation is not determined, but it is very small. Mortality stated to be about 75%, but figure probably unnecessarily high, and capable of reduction with adequate treatment.

Action.—The cyanids stop the gaseous interchanges between blood and tissue cells and therefore induce tissue asphyxia. There is also a short period of stimulation of the motor centers of the nervous system, followed by their paralysis. Death usually takes place within one hour, maybe in a few minutes, and if death is not early recovery is to be expected. The fact that the heart is not directly injured, but merely has its action restrained by interference with its cellular oxidations, increases the likelihood of success in resuscitation if artificial respiration is persisted in.

Symptoms.—When taken by mouth,—

Taste, hot, bitter, acid ; sense of constriction in throat ;
Sense of compression of head, within a few minutes ;
Vertigo, loss of consciousness, the victim
Falling in a convulsion, jaws set and fists clenched.
The respirations are stertorous, the pulse fast and weak.

If vomiting occurs now recovery is likely. Otherwise
Convulsions, with perhaps opisthotonus, set in for a time before

Paralysis of the motor centers develops.

Inspirations are little gasps ; expirations, prolonged and followed by a pause before the next inspiration follows.

Asphyxia causes death unless managed by the attendants.

When an overwhelming dose is taken in a readily absorbed form

An almost immediate convulsion follows the draught, the victim

Falling, then lying still, the face bloated, eyes staring, foam at mouth, involuntary evacuations of bowels and bladder taking place,

The skin becomes cold and clammy and the muscles gradually relax.

Short gasping inspirations are made, but death is likely to take place before aid can be given unless vomiting occurs. **When the gas is inhaled in large quantity**, there are

Faintness, dizziness, muscular twitchings, the victim soon

Falling unconscious in convulsions, the termination being much as in the preceding cases.

Treatment.—If drug taken by mouth, empty stomach and perform lavage with potassium permanganate solution, 10 grains to the pint. Even if patient appears dead or hopelessly stricken, artificial respiration with oxygen inhalations should be persistently applied. The cardiac cessation and the collapse are of such a nature that vigorous stimulation can be used with safety and advantage,—cold water douche to spine, neck and head; ice rub, alternated with hot compresses to chest; cold towel rub alternated with fomentations. But artificial respiration continuously.

Nitrobenzene, or
Oil of Mirbane, or
Artificial Oil of Bitter Almond.

Source.—Most frequently accidental, in connection with its industrial uses. Occasional deaths have followed when persons have tasted the substance not realizing its poisonous nature, when they found it in perfumes, or flavoring extracts. A few cases of suicide are recorded.

Fatal Dose.—If taken by mouth and not vomited, probably about 15 minims ($\frac{1}{4}$ teaspoonful), altho this may be recovered from. Very large quantities have been recovered from when vomiting took place. When absorbed by the lungs from its fumes in the air, the quantity required cannot be stated.

Action.—Pronounced power to form methemoglobin in the blood and to thus check tissue oxidations, therefore resembling the cyanids. Also mildly stimulates, then depresses central nervous system.

Symptoms.—Discoloration of lips, other mucous membranes or skin is usually the first sign, this due to the destruction of the erythrocytes of the blood. Cyanosis develops. The odor of the substance, resembling that of peach pits or bitter almonds, is almost always appreciable about the person.

Headache, and a sense of anxiety; giddiness,
Nausea and vomiting appear. Unless the case be one of severity,
Convulsive twitchings of the muscles take place and
Involuntary snapping of the jaws. In more severe cases there is soon

Rapidly developing coma accompanied by
Weakening of pulse, respiration, sphincter control,
and

Lowering of body temperature.

Cyanosis is marked thruout.

Even from this condition of coma spontaneous
recovery may take place.

Death is unlikely if any remission in the coma can
be secured.

Treatment.—If the poison has been swallowed, washing out
the stomach is of first importance, if it can be
done without seriously aggravating the cyanosis.
If stomach tube not used administer an emetic.

Artificial respiration and oxygen inhalation are the
measures most depended upon after the condition
has become advanced. Even before artificial re-
spiration is necessary oxygen inhalation seems
of value.

Other measures for managing the condition will be
suggested by the symptoms. The anxiety con-
tributing to shock or mental excitement may be
controlled by morphin or chloroform if seems
necessary.

Blood-transfusion, or venesection, followed by com-
pound saline infusion (see Collapse and Acidosis)
is recommended in desperate cases.

GROUP IV.

Respiratory Irritation or Asphyxiation.

In this group may be included all gaseous poisons, tho they act in a variety of ways. The following grouping of these substances will help emphasize their mode of action.

- (a) **Causing death by injury to respiratory passages,—**
Ammonia gas,
Bromin,
Chlorin,
Formaldehyde,
Mineral acid fumes.
- (b) **Causing death by displacing oxygen from the lungs,—**
Carbon dioxid.
- (c) **Causing death by alteration of the blood or by action after absorption,—**
Anilin fumes,
Carbon monoxid,
Hydrogen sulphid,
Hydrocyanic acid gas,
Nitrobenzene,
Nitrogen monoxid, or "laughing gas."

In sub-group (a) the symptoms are lacrymation, coughing becoming more and more violent, dyspnea, suffocation from spasm of the glottis. Pneumonitis is a frequent sequel, when primary recovery has taken place.

In sub-group (c) the symptoms are not so constant. There may be almost instant death if the concentration of the gas in the inspired air be high, or a period of hours may be required for the symptoms to develop.

The treatment in every case includes the removal of the patient to fresh air and the employment of measures to establish and maintain oxygenation of the blood.

Ammonia Gas.

Cases of poisoning most frequent about gas works, tho occasional in many different industrial plants.

Altho death is almost always, when it occurs, due to spasm of the glottis, there is a possible absorption which, causing irritation of the central nervous system, results in convulsions or muscular twitchings. The usual lacrymation, coughing, dyspnea, strangulation and suffocation occur. Pneumonitis is an after effect.

Treatment.—Remove patient to the fresh air.

Give for inhalation steam vapor or **vapor from very dilute acetic acid** or vinegar, as chemical antidote.

If spasm of the glottis has occurred perform **intubation or tracheotomy**, and if necessary give artificial respiration with oxygen.

Anilin.

Cases of poisoning almost limited to manufacturing chemical plants where this substance is employed. Not of frequent occurrence.

Action is partially due to depression of the central nervous system, but largely due to alteration of the hemoglobin of the blood to methemoglobin. There are but very mild irritative symptoms.

The symptoms begin with a grey-blue discoloration of the lips and mucous membranes, usually before the patient feels ill. Headache, nausea, vomiting, dizziness, labored breathing, muscular twitchings, unconsciousness, convulsions, and death appear in order as the poisoning advances.

Treatment.—Remove patient to fresh air.

Give oxygen inhalation and, if needed, artificial respiration.

Meet dangerous symptoms as they occur.

Bromin.

Cases occur almost exclusively in chemical factories and laboratories.

The action of this substance is so intense that 1 part in 10,000 in air, if breathed for a period of a few hours, may cause death from ulceration of the respiratory mucous membranes and the later pneumonitis.

The symptoms and treatment are identical with those of ammonia with the exception of the use of **dilute ammonia vapors for inhalation** as a chemical antidote instead of dilute acetic acid vapors. A spray of dilute solution of sodium bicarbonate may reach and neutralize some of the irritant in the upper portions of the respiratory pathway.

Chlorin.

(See Bromin, preceding.)

Carbon Dioxid.

Most frequent occurrences in connection with fires, in coal mines, breweries and wineries.

This gas is not highly poisonous, its only action in the blood being to interfere with oxygen conveyance and to increase the irritability of certain nerve centers. Its danger comes from its exclusion of oxygen from the lungs. Slow poisoning by this gas is rare.

To produce asphyxia close to 10% of carbon dioxid in the inspired air is necessary. This can be estimated by the marked dimming of the flame or the extinguishing of a candle by an air containing this quantity.

Symptoms.—Reddening of face, tingling of body, sense of irritation of respiratory mucous membranes, dyspnea, palpitation of the heart, unconsciousness, death unless rescued.

Treatment.—Remove patient to fresh air, being careful that rescuers be not permitted to be overcome by the gas.

Give oxygen inhalation and artificial respiration. Pulmotor or Lungmotor ideally suited to such cases.

Success should be expected in cases even tho the patient may have been unconscious for some length of time.

Carbon Monoxid, Illuminating Gas.

Source.—This is one of the most frequent causes of death from poisoning, there being a very large number of deaths annually from the suicidal and accidental inhalation of illuminating gas. There are accidental cases also about blast furnaces, smelters, coal mines, and from the incomplete combustion of carbon in any of its forms, whether in gas-burners or coal stoves.

Action.—Carbon monoxid has a very high affinity for hemoglobin and is very rapidly absorbed from the pulmonary alveoli by the blood, with the formation of stable carboxy-hemoglobin. This prevents the conveyance of oxygen by the blood and excites tissue asphyxia. The blood assumes a permanent cherry red color. There appears to be an additional direct paralysis of the nerve centers.

So small a quantity as 3 parts in 1000, if breathed for a period of a few hours, may result in death. If the quantity in the air is large the patient may drop unconscious with the first few breaths because of immediate asphyxia of the heart muscle, death occurring after a few abortive attempts to breathe.

Symptoms.—When the proportion in the air is small,—

Headache, dizziness, malaise, drowsiness, nausea, vomiting (sometimes a period of transient excitement), unconsciousness, convulsions, shallow respiratory movements, failing pulse, death. Red blotches upon the skin frequently seen.

Treatment.—Results uncertain, because of chemical alteration of the blood.

Oxygen inhalation and artificial respiration.

Transfusion of blood. Symptomatic treatment for other conditions.

Sequelae.—Persisting headache, dizziness and nausea, even when rescued early.

If poisoning reached a fairly advanced stage, a most varied set of nervous system symptoms may follow,—peripheral neuritis, paralyses, melancholia, or mania.

Formaldehyde Gas.

Symptoms and treatment the same as for ammonia gas, excepting the substitution of very dilute ammonia vapor for acetic acid vapors for inhalation.

Hydrocyanic Acid, see Group III.

Hydrogen Sulphid.

This is a relatively rare form of poisoning. It is set up sometimes by sewer gases, and there are numerous industrial processes where it might cause poisoning were precautions not taken to prevent it.

Its action is dependent upon its vigorous affinity for the hemoglobin and alkaline elements of the blood, which results in hemolysis and darkening of the blood. There is also a mod-

erate irritating action upon the respiratory mucous membranes and a paralyzing action upon the central nervous system. The irritant effects upon the mucous membranes are due to the corrosion following the formation of alkali sulphids by the interaction of the gas with the alkaline secretion of the respiratory tract. These irritations are but slowly recovered from. As with carbon monoxid, inspiration of the concentrated gas may cause the person to fall dead without the development of any symptoms.

Symptoms and Treatment, same as for Carbon Monoxid, preceding, except for greater respiratory irritation and somewhat increased tendency to convulsions.

Nitrobenzene.

See Nitrobenzene at end of preceding Group III.

Nitrous Oxid, "Laughing Gas."

Cases encountered only as complications of intentional anesthesia; therefore discussion not required in this subject.

SUMMARY OF USES OF ANTIDOTES.

Chemical Antidotes.

"General Antidote".—

Uses.—When no diagnosis has been made.

Mix to a paste 1 tablespoonful of magnesia and 2 tablespoonfuls of powdered charcoal; add to a pint of water; administer in half-tumbler doses and after last dose empty stomach.

Albumin, see Egg White.

Alcohol (Grain).—

Uses.—Strong Carbolic Acid, Lysol, Creolin, etc.

Dilute alcohol with 4 volumes of water; administer a tumbler full; promptly remove from stomach either by emesis or stomach tube. If possible use this solution for thoro lavage.

Ammonia Water.—

Uses.—Formaldehyde solution.

Dilute ammonia water until its vapors can be inhaled comfortably; give one pint; use freely for lavage.

Calcium Carbonate, powdered chalk, marble dust, powdered wall plaster.—

Uses.—Oxalic Acid, Sulphuric Acid, Hydrochloric Acid, Nitric Acid, etc.

Mix 2 teaspoonfuls with a glass $\frac{2}{3}$ full of water for each dose. Repeat until neutralization seems to have been accomplished. This substance is inferior in general to Magnesia.

Calcium Chlorid.—

Uses.—Oxalic Acid or soluble salts of oxalic acid.

Dissolve 1 to 3 teaspoonfuls in a glass of water, according to the amount of poison taken and administer at once. It is well to remove from stomach after reaction has taken place.

Also, add 1 gram (15 grains) to 500 cc. of compound saline intravenous infusion and administer to replace calcium removed by the absorption of oxalic acid.

Calcium Lactate.—

Same as for Calcium Chlorid, and much preferable if obtainable.

Citric Acid, or strong lemonade.—

Uses.—Sodium, Potassium, or Ammonium Hydroxids, Sodium Carbonate, etc.

Dissolve 1 tablespoonful in a quart of water and administer until evacuated stomach contents have an acid reaction.

Copper Sulphate.—

Uses.—Phosphorus, as an antidote; other times as emetic.

Dissolve 16 grains (1 gram) in a pint of water and give half-glassful doses every ten minutes until vomiting.

Or empty stomach with tube if no vomiting without it when the full pint has been given.

Egg White or Albumin.—

Uses.—Mercury, Formaldehyde solution, Lead, Copper, other metallic salts, Mineral Acids, Carbolic Acid when alcohol not available, and as a demulcent.

Mix the whites of eggs with a little water and administer freely. In case of mercury and carbolic acid poisonings it is very important to remove the resulting albuminate from the stomach, for it is absorbable, tho slowly so.

Ferric Hydroxid with Magnesia.—

Uses.—Arsenic. (See Arsenic, Treatment, p. 57.)

Iron, Dialysed.—

Uses.—Arsenic. (See Arsenic, Treatment, p. 57.)

Inferior to the preceding.

Lime Water.—

Uses.—Oxalic Acid, oxalates, any acid.

Administer freely. Disadvantage if there is much acid to be neutralized, because it is so dilute.

Magnesium Oxide, or Magnesia.—

Uses.—Acids of all kinds; fair for Arsenic, Mercury, Phosphorus, and other irritant metallics.

Mix a tablespoonful into a glass of water and administer.

Such a dose may be repeated several times if needed.

Magnesium Sulphate, or Epsom Salt.—

Uses.—Barium Salts, Lead Salts, and as cathartic.

Dissolve 1 tablespoonful in glass of water and administer.

May be repeated as desired,

Milk of Magnesia, Magnesium Hydroxide.—

Uses.—Same as Magnesia.

Two to 4 ounces, mixed with water or milk and repeated as needed.

Milk.—

Uses.—All acids, metallic salts, Phenol and Formaldehyde solution. Is less efficient than many of the others but very soothing.

Administer freely. In poisonings where oils are to be avoided, the milk should be skimmed (as in Phosphorus, Copper Salts, Cantharides, the volatile oils).

Potassium Ferro-cyanide.—

Uses.—Copper Sulphate.

Dissolve 1 tablespoonful in a pint of water and use for lavage.

Potassium Permanganate.—

Uses.—Phosphorus, Hydrocyanic Acid, all alkaloids.

Dissolve $\frac{1}{2}$ to 1 dram in a gallon of water and use for lavage.

Soap: Castile, Ivory, or other good quality, unmedicated soap.—

Uses.—For corrosive acids and all metallic salts.

Dissolve shavings of soap in four or five times their bulk of warm water. Administer freely.

Sodium Bicarbonate, or Baking Soda.—

Uses.—In acidosis, for zinc salts and mineral acids (not Oxalic).

Rapid liberation of carbon dioxide from this salt makes it dangerous for use against the strong acids; solubility of sodium oxalate prohibits use in oxalate poisoning.

In acidosis, intravenously or by mouth (see Acidosis). For zinc salts, 1 dram or more (according to amount of poison to be precipitated) dissolved in water.

Sodium Carbonate, Washing Soda.—

Same as for Sodium Bicarbonate, but more irritant.

Sodium Chlorid, or Table Salt.—

Uses.—Silver salts; to augment action of dialysed iron in arsenic.

Dissolved in water used freely as needed.

Sodium Phosphite. —

Uses.—Corrosive Sublimate.

Secured in prepared form from Abbott Alkaloidal Co.

Sodium Sulphate, or Glauber Salt.—

Uses.—Barium or Lead salts; Carbolic Acid.

Dissolved in water, in two-teaspoonful doses to effect. 1% added to intravenous infusion, not more than 5 gm. in 12 hours, to replace sulphates removed by carbolic acid.

Tannin; Strong Tea or Coffee.—

Uses.—Antimony, colchicum, and all alkaloids (potassium permanganate seems somewhat better for this latter class); fair precipitant action on all metallic salts, but quantity needed for these too large to be employed conveniently.

Dissolve 1 dram in a pint of water and administer, or use for lavage.

Turpentine, Old Oil of.—

Uses.—Phosphorus.

$\frac{1}{2}$ dram emulsified in 1 ounce mucilage of acacia; dose repeated every 30 minutes until four doses taken (acts as a combined carthartic and antidote).

Vinegar.—

Uses.—Alkali poisonings; Ammonia vapor.

Dilute with about three volumes of water and give to drink sufficient to neutralize the poison.

Inhalation of vapors from boiling dilute vinegar after ammonia vapor poisoning.

Physiological Antidotes.

(Frequently recommended, but not always rational.)

Adrenalin, in intravenous infusion, 1 cc. to litre, slowly given.

Atropin, $\frac{1}{150}$ gr., repeated,— for lead colic, vagus cramps, vagus heart slowness, vagus asthmatic breathing.

Caffein, as tea or coffee, by mouth or enema, or with sodium benzoate hypodermically (2 gr.),—for general or medullary stimulation and for digitalis heart block.

Chloroform, by inhalation, to control strychnine or other severe convulsions. Usually considered less safe than ether.

Ether, same as chloroform, but preferable.

Morphin, $\frac{1}{8}$ to $\frac{1}{2}$ grain, to control pain and excessive mental excitement.

Nitroglycerin, $\frac{1}{200}$ gr., repeated two to four times, to reduce blood-pressure.

Oxygen, by inhalation, in respiratory failure or cyanosis.

Pilocarpin, $\frac{1}{4}$ gr., to aid elimination, particularly of wood alcohol.

Pituitrin, 1 ampoule, in some types of collapse.

Sodium Bromide, $1\frac{1}{2}$ drams, in solution, by rectum or mouth, to reduce convulsions; smaller dose for delirium.

Strychnin, $\frac{1}{40}$ to $\frac{1}{20}$ gr., as general stimulant in depression.

Saline Infusion (see Collapse).

All of the foregoing must be used with discrimination; the less they are resorted to the better.

SECTION II.

CHRONIC AND CUMULATIVE POISONING.

It will be remembered that the distinction between these two types lies in the gradual development of the symptoms of chronic poisoning and their sudden development in the cumulative type.

The most important instances of chronic poisoning arise in connection with the certain industrial processes in which poisonous substances are employed. The number of substances which may influence the health of those persons who habitually handle them is very large. Particularly among employees of chemical manufacturing concerns there are likely to be found a great variety of disturbances of health due to the conditions of their work. In most of these cases, the worker himself appreciates the poisonous nature of the substances with which he deals and, to as great a degree as possible, takes precautions to avoid injury by the more active chemical poisons; so it is only those whose effects develop slowly and insidiously that the physician must bear in mind as the possible unsuspected causes of ill health when his patient suspicions merely a chronic disease of some nature.

Every physician in the routine examination of patients should make it a rule to determine, not only the present, but the previous occupation of the patient. Knowing, then, those occupations in connection with which certain types of intoxications frequently appear, the physician will be in a position at once to know the likelihood of exposure to the various types of intoxication that might occur.

In addition to these cases of industrial poisoning which it should be relatively easy to detect, there are two other modes of occurrence of chronic poisoning. (1) That due to long continued administration for therapeutic purposes of drugs which are relatively difficult of elimination, or which, because of certain agreeable sensations produced by their action, lead to their continued use beyond the time of their need. (2) That due to the criminal, secret administration of a drug with intent to kill. This latter form of chronic poisoning occurs nowadays only with great infrequency, and is usually one of a relatively small list of rather common poisons, namely: arsenic, antimony, mercury, phosphorus, lead. The appearance of the symptoms of chronic poisoning by any one of these, in the absence of knowledge on the part of the patient of how any of them might have been administered, may be permitted to arouse suspicion of either criminal poisoning or accidental poisoning through food or air. The physician under such circumstances must settle the question by careful study

of the patient's environment and by first requiring that all foods and drinks ingested by the patient be secured from a source of proven dependability; or second, by requiring that the patient be in continual charge of some person known to the physician to be absolutely trustworthy. Disappearance of the symptoms will strongly indicate a previous poisoning. Cases requiring this procedure will be very infrequent, as homicidal poisoning and poisoning other than bacterial in nature, thru foods are becoming fewer every year.

The important poisons which produce chronic symptoms through excessive therapeutic use are: calcium, camphor, ether, chloroform, alcohol, chloral hydrate, bromides, morphine, heroin, cocaine, antipyrin, acetanilid, quinine, salicylic acid, mercury, lead, bismuth, arsenic. In case of some of these there is little likelihood of the patient himself having continued the use of the drug without some physician's orders. Some are likely to produce chronic symptoms only during the course of administration of the drug, while still others produce effects which can be noted for a considerable period of time after discontinuing the drug. Perhaps the most important of these are the ones which bring about the formation of a habit.

The poisons which are most frequently causes of chronic industrial poisoning, together with the occupations in which the poisons are likely to occur, are as follows:

Lead	Paint manufacturers, painters, pottery workers, file cutters, type setters, linotypers, printers, workers in zinc smelters, electric storage battery plants, and dye factories.
Phosphorus	Match workers, manufacturing chemists.
Arsenic	Paint makers, furriers, taxidermists, undertakers, laborers about smelters, and sulphuric acid factories.
Mercury	Workers about incandescent electric lamp factories, in barometer and thermometer making, in felt factories, and certain metallurgical plants.
Carbon monoxide	Workers about gas factories, coke plants, blast furnaces, cement works, coal mines, and others.
Copper and Zinc	Machinists and brass founders.

Nitrogen derivatives of benzine, and other nitro compounds,—as nitroglycerine and anilin Nitroglycerine workers, chemists, anilin dye workers.

The treatment of the chronic poisonings consists in (1) preventing any further administration of the drug, or in case of habit-forming drugs gradually withdrawing them if it seems better. (2) Accelerating, if possible, the elimination of the drug from the patient's system. (3) Relieving the patient from the distressing symptoms of the disturbance as much as possible. (1) in industrial poisoning is a matter of hygienic factory management; (2) and (3) are the duties of the physician. The relief of symptoms which need relief is a problem for the physician to solve on the basis of his general training. In case of the habit-forming drugs, the stoppage of the drug is one of the biggest of the physician's problems, and is a subject larger than can be undertaken here. The assistance in the elimination of the accumulated drug from the patient's body is the phase of the question which can be briefly mentioned here.

The methods to be followed in the elimination of the poison from the patient's body will to some extent be determined by the nature of the substance. There are some which are supposed to be specifically eliminated by definite medicinal administrations, and these will be spoken of in connection with each substance in the following list. The general procedure to be followed, however, is the application of all measures for increasing the activity of elimination thru the skin and intestines, as well as the kidney, and to overcome passive congestions of the liver, which is so often the place of accumulation of the excess of the drug. Remembering also that these chronic and cumulative poisonings occur predominantly in cases where kidney elimination is deficient, either because of the action of the drug or because of a pre-existing nephritis, it will be evident that those measures of augmenting elimination which do not irritate the kidneys, but which rather utilize the vicarious modes of elimination are the rational ones to utilize. Hence the diaphoretic measures cited in Section I will be of prime importance; and with these osteopathic manipulations as indicated will be found of distinct assistance.

Following is a list of the symptoms characteristic of the more important forms of chronic and cumulative poisoning:

CAUSING CHRONIC TYPE OF POISONING.

Acetanilid.

Digestive irritability, neuralgic pains, cutaneous irritability with recurring rashes, itching of the skin, cardiac depression causing dyspnea on mild exertion, cyanosis, methemoglobinemia. On withdrawal of the drug, headache, nervousness, irritability.

Anilin.

Greyish-blue mucous membranes and peculiar greyish alteration of color of skin sometimes before any other symptoms evident. Tendency to headaches, and malaise, easy production of dyspnea, digestive disturbances, gradually developing albuminuria, tendency to chronic cystitis (tumors of the urinary bladder reported frequent among anilin workers), anemia due to methemoglobinemia.

Antipyrin.

Practically identical symptoms as shown by acetanilid, but cyanosis and methemoglobinemia do not occur, and therefore the other manifestations are less pronounced.

Alcohol.

Important because of its habit-forming properties. Gastro-intestinal irritability; chronic catarrhal gastritis with hyperchlorhydria in earlier stages; injected venules of nose, cheeks, and conjunctivae; trembling of hands and lips; restlessness, nervous irritability, lack of self-control; peripheral neuritis, gradually increasing in severity; loss of memory combined with tendency to "lose one's bearings" (Korsakoff's psychosis).

Arsenic.

Reported to be a habit-drug in some parts of the world, because of its production of a mild degree of euphoria and aggressiveness. A source of accidental poisoning because of its occasional presence in the coloring matters used on wall paper, artificial flowers, colored rubber toys, various forms of enamel, etc.

Marked gastro-intestinal disturbances, swollen and tender liver, peripheral neuritis, cutaneous eruptions of various kinds, edema particularly affecting the face, pigmentation of the skin, "arsenic pock," conjunctivitis where poisoning from vapors containing the drug.

Potassium iodid recommended to assist elimination, but use not experimentally justified.

Antimony.

Occurs occasionally among typesetters.

May be mistaken for plumbism. Constipation, insomnia, anemia, mild neuritis, nervous irritability.

Bismuth.

Usually due to absorption from surgical packing of sinuses. Salivation, stomatitis, blue line on gums, gastro-enteritic disturbances, colic, nephritis slowly developing.

The Bromids.

Mental dullness tho irritable, poor memory, physical inactivity, gastritis, poor appetite, constipation, varied cutaneous eruptions including pustular forms.

Elimination assisted by liberal use of sodium chlorid and water.

Caffein.

Of importance because of its wide use in the form of tea, coffee, and soft drinks, as Coca Cola.

Insomnia, and irritability of the cardiac muscle, both of which may seriously aggravate the condition of a separate defect of the cardiac mechanism. Missing the usual stimulant influence of the drug causes appearance of marked nervous irritability, headache and aggravation of the heart symptoms, until abstinence becomes the habit.

Calcium Chlorid, or Lactate.

Constipation, tingling or numbness of arms, hands and legs, tinitus aurum, sometimes venous thrombosis.

Camphor.

An occasional habit drug. Could usually be recognized by presence of odor on patient's person. Symptoms those of mild cocainism.

Carbon Monoxid.

Causes a secondary anemia with all accompanying symptoms, with an additional weakness of limbs and tendency to headache.

Chloral Hydrate.

Mental sluggishness, physical weakness, gastro-intestinal disturbances, constipation, anemia, cutaneous eruptions.

Sodium bicarbonate as an alkalinizing agent is believed to aid in elimination.

Chloroform.

An occasional habit drug. Symptoms same as chloral, but less gastro-intestinal irritation.

Cocain.

In absence of the usual dose, or when given a placebo in place of it, nervous irritability, muscular twitchings, insomnia, moroseness. Physical condition displays gastro-intestinal disturbances, loss of appetite, constipation, anemia, loss of weight, neglect of the duties of everyday life, termination in chronic dementia or mania.

Local indications at point of administration,—minute, bluish scars of needle punctures; ulceration of nasal mucous membrane; gingivitis.

Atropin used, while withdrawing the drug, to supply some cerebral excitation by a non-habit-forming drug.

Copper.

Brass-founders' Ague produced by brass dust. (See Zinc.)

Chronic irritation of gastro-intestinal tract when taken over long periods of time in foods.

Heroin.

Practically the same as Morphin, but less somnolence.

Hydrogen Sulphid.

Irritation of mucous membranes (lacrymation), marked enteritis, anemia, lassitude. The symptoms tend to persist after removal of the patient from the influence of the gas.

Lead.

The type symptoms are Colic, Palsy, Encephalopathy.

There is sweetish taste in the mouth, diminished salivary secretion, a blue line on the gums, colic, constipation, anemia, elevated blood-pressure, wrist drop, persistent headache, rarely acute attacks of mania.

Mercury.

Salivation; soft, swollen gums; no dark line on teeth; loosening of teeth; fetid breath; sore throat; derangements of digestion; swollen and painful joints; mild cachexia; muscular tremors; irritability of temper; sometimes a recognizable peripheral neuritis. Potassium iodid to aid elimination and potassium chlorate for stomatitis are recommended.

Morphin, and other Opium Derivatives.

Without usual dose patient is restless, irritable and unable to concentrate his attention.

When under the influence, quiet, self-satisfied, or somnolent.

Systemic condition shows digestive disturbances with marked constipation; loss of appetite, loss of weight; mental depression, with irritability; headaches; anemia; polyuria and sometimes glycosuria; amenorrhea; and impotence, sometimes; final tendency toward mental and moral depravity. Hyoscin and atropin used in beginning treatment of morphinism.

Phenol.

Usually from surgical dressings.

Gradually developing respiratory and circulatory weakness; albuminuria; absence of sulphates from urine; stupor.

Phosphorus.

Swollen gums, pain in jawbone, followed by pyorrhea, and inflammation of the jawbone, terminating in necrosis. Also after absorption of drug, development of anemia, albuminuria, sometimes jaundice, gastric disturbances, sterility in females, and sometimes cachexia.

Sodium bicarbonate of value in the systemic treatment.

Quinine.

Condition developed designated cinchonism. Sense of fulness in head and ringing in the ears; muscular weakness, and unsteady gait; mental dulness; disordered vision; deafness; slow, weak pulse; gastro-intestinal disturbances; skin eruptions.

Salicylic Acid and its Salts.

Salicylism, the common effect of over-use, long continued, is practically identical with cinchonism.

Salicylic intoxication sometimes develops after the manner of a cumulative poisoning (see below).

Zinc Compounds.

The cause of "brass-founders' ague" (see in next list).

CAUSING THE CUMULATIVE TYPE OF POISONING.

Drugs, which are slowly eliminated, which display a narrow margin between the safe and the dangerous dosage, and which (as general protoplasmic poisons) may cause a nephritic condition which would progressively retard elimination, sometimes tend to produce suddenly developing symptoms during the course of the routine administration of the drug. Certain other conditions, such as variation of the patient's condition apart from any influence of the drug, may be responsible for such manifestations. The drugs most frequently producing such, so-called, cumulative action are the following:

Arsenic.

Puffiness appearing beneath the eyes; diarrhea with colic; catarrhal rhinitis with lacrymation; headache; albuminuria; neuritic pains.

Bismuth.

Symptoms given under chronic type may appear suddenly and resemble cumulative action.

The Bromids.

The erythemas may develop suddenly and resemble some acute exanthem.

Cocain.

Sometimes after local application for anesthetic purposes, some very slight occurrence may excite distinct cocain collapse; or a period of mental excitement may develop.

Not a true cumulative action.

Colchicum.

Nausea, vomiting, diarrhea, and albuminuria may suddenly be developed in the course of a gout treatment.

Digitalis and its Preparations.

Loss of appetite, nausea, vomiting, diarrhea; cardiac anxiety, palpitation, missed beats, nodal rhythm; headache.

The cardiac disturbances are most varied, and require close study to determine the exact nature of the condition that has been set up.

Strophanthus preparations in the management of the condition merely replace the heart tonic effects of the digitalis and do not act to overcome its effect, as some workers carelessly assert.

Nitroglycerin, or the Nitrites.

Sudden onset of pounding of heart, flushing of face and neck, sense of fullness in head and frontal headache. Usually these symptoms are of gradual development, or may result from a single excessive dose.

Quinine.

The varied forms of cutaneous eruptions may appear suddenly without the other manifestations of cinchonism having developed.

Salicylic Acid.

Salicylic intoxication,—talkativeness, exciting hallucinations, desire to keep continually active muscularly, and to get out of bed if confined.

Sulphonal.

In nephritic patients where being used for insomnia,—nausea, vomiting, stupor interrupted by hallucinations.

Zinc.

Due essentially to inhalation of zinc oxid fumes. There are periodic attacks characterized by irritable cough, malaise, nausea, headache, muscular tremors developing to distinct rigor, rise of body temperature, asthmatic breathing and palpitation of the heart. The attack terminates with a profuse sweat, and the worker is usually ready for work the day following the commencement of the attack. This attack is popularly designated "Brass-founders' Ague."

SECTION III.

THE DUTIES OF THE PHYSICIAN IN CASES OF POISONING.

(With frequent paraphrasings from Witthaus.)

Primarily, the duty of a physician when called to a case which is found to be one of poisoning, is to

(1) Rescue the patient from the effects of the poison or corrosive already taken.

All of the preceding pages of this manual have concerned themselves with the discussion of the performance of this duty. But not all cases of poisoning are accidental or self-inflicted, a small percentage of them are attempts at murder. In these cases it devolves upon the physician to stand between the community and the poisoner, as the protector of the one or the other; for it is largely within the hands of the physician, in case of death, by signing a death certificate without proper investigation, to permit the loss of the evidence, the body of the victim, upon which the proof of the crime depends. We may therefore state two more duties of the physician in cases where the origin of the poisoning is obscure:

(2) To prevent any further administration;

(3) In case of death, to avoid interfering with a proper investigation by giving a certificate of death or permitting the undertaker to embalm the body.

To carry out the second duty it is necessary for the physician to assume, to a certain extent, the unpleasant duty of acting as a detective, using the greatest care, however, to avoid throwing suspicion upon any individual until there is adequate ground for it. Therefore, in the management of cases of poisoning where there is any question as to the nature of the poison, or the manner whereby the patient secured it, these rules should be observed:

(1) Prevent the destruction or disposal of the vomitus and urine from the patient, having it collected for examination in the chemical laboratory, and keeping it in such a manner that any tampering with it could be recognized. Any medicine recently used by the patient, and any food of unusual or suspicious nature should be similarly attached by the physician for examination.

(2) If there is any suspicious circumstance attending the case, or any person among the attendants whose actions arouse suspicion, the physician should provide a means of isolation of

the patient from everyone excepting a person known to him and who has no non-professional relationship toward the patient,—a special nurse, for example.

(3) A careful case record of all poison cases should be kept.

(4) After death refuse to sign any death certificate until an autopsy has been secured, with the co-operation of the proper authorities.

(5) In case of finding the patient dead at arrival at the case, written notes should be made of the time, place, position of the body, names and addresses of those present, the condition of the clothes, evidences of vomiting, disposition of the material, appearance of "fixing up" of the body before arrival; description of any cup, bottle, vessel, weapon, or paper near the body and its relation to the body. These may be needed should the physician be called as a witness in connection with any legal proceeding concerning the individual's death, tho the case be suicidal, homicidal, or accidental.

It is not to be presumed that the physician should carry out any investigation. It is simply his duty to see that it is made possible for an accurate investigation to be carried out.

When a death certificate has been refused, pending the performance of an autopsy, he should call for assistance upon one especially trained in this branch of work. "The practitioner of medicine, not especially engaged in pathological investigation, should avoid assuming the responsibilities of a medico-legal autopsy. Indeed, even coroner's physicians are more frequently found ignorant than informed of the details which should be regarded in an autopsy after death supposed to be due to poison. Whenever it is possible the postmortem examination should be conducted by a skilled and experienced pathologist, and the chemist who is to make the analysis should be present. * * * The peculiar experience and training requisite to enable one properly to conduct an intricate toxicological analysis are such that only exceptionally can persons truly competent be found, except among those who in the larger institutions of medical and pharmaceutical instruction have devoted themselves specially to this subject."

That a general practitioner should assume that he could participate in such an investigation is truly amusing to one realizing the difficulties involved. We will therefore dispose of this topic with the mere statement that careful observations are made concerning all of the gross aspects of the cadaver, external and internal, as in any well-conducted autopsy; after which, the various organs, the stomach and intestines each separately removed with their severed ends ligatured to retain their contents, the residual urine in the bladder, the fluid ex-

update in the body cavities, are individually placed in proper receptacles for proper preservation and protection until they can be minutely examined chemically and microscopically.

The objects to be attained by a proper examination may be stated (1) to determine the possibility of other cause for death than poisoning; and

(2) To secure data, chemical and pathological, which even in the absence of isolation of the poison, would lend support to other matters of evidence bearing upon the case.

Of course, it is hoped, in case of actual poisoning, to isolate the chemical substance which was responsible for death. In numerous instances this is utterly impossible. When it is isolated from portions of the body where its presence proves widespread distribution of the poison, there is established very powerful evidence of the exact cause of death. This "poison, * * * presented to the judge and jury in a permanently stable form, capable of impressing the senses," is, according to Brundage and some other toxicologists, called the "Corpus Delicti." It is supposed to be some of the identical drug that was criminally used to cause the death of the victim, secured from his dead body. Altho this meaning of the term is not correct in a legal sense, it is in such wide use among the rank and file of the toxicologists of the country that the student should make note of it.

The detail of the establishment of the causation of a death by a specific poison is too complex a matter to be dealt with here. If the physician will follow the suggestions made above, and promptly communicate with proper officials when a situation develops of which he is not master, he will have performed his duty.

ADDENDA.

THE CHEMICAL DETECTION OF POISONS.

This is not a subject with which the physician need concern himself. It is impossible for anyone other than a specialist in this branch of medical chemistry to attain any competence in the work. Therefore there will be included here merely an excerpt from Autenrieth, "Detection of Poisons," giving in outline the general procedure followed in the examination of a suspected specimen; and, because of the frequent reference to it, a description of Marsh's Test for arsenic, in solution. It is suggested that, when a physician is required by anyone to tell about the detection of poisons, he inform the person of the highly special nature, both of his training and that of the man who would be prepared to discuss such a question, and refuse to commit himself to any inaccurate statement, such as he would almost have otherwise to make.

Outline of Procedure.

Nearly all the common poisons and drugs may be placed in one of three groups. This classification, based upon the chemical behavior of these substances during isolation from mixtures, is as follows:

Group I.—The members of this group volatilize without decomposition when heated and distil from an acid solution with steam. Yellow phosphorus, hydrocyanic acid, carbolic acid, chloroform, chloral hydrate, iodoform, aniline, nitrobenzene, carbon disulphide and alcohol are the principal substances of this class.

Group II.—The members of this group are non-volatile, organic substances which do not distil from an acid solution with steam. But hot alcohol containing tartaric acid will extract them from extraneous matter. Alkaloids, many glucosides and bitter principles, as well as certain synthetic organic drugs like acetanilide, phenacetine, antipyrine, pyramidone, sulphonal and veronal comprise this group.

Group III.—This group includes all poisonous metals.

In toxicological analysis, therefore, poisons are divided into three groups, each of which has its own special methods of procedure. A few poisons like mineral acids, caustic alkalies, oxalic acid and potassium chlorate cannot be conveniently placed in these three groups owing to differences in solubility and other peculiarities. Special tests for such substances must be made with a separate portion of material.

The material must be thoroughly mixed and divided into three or four approximately equal portions, unless the analysis is to be limited to the detection of a single well-defined substance. One portion is tested for non-volatile, organic substances. The second portion is examined for volatile poisons and the residue from this portion is used in testing for poisonous metals. The third portion is tested for substances (which cannot be placed in any of the three main groups and which require specific tests). The fourth portion is held in reserve in case additional material is needed to verify a doubtful result, or to replace a portion accidentally lost during analysis.

Occasionally it is advisable to depart from the general procedure and follow a special method, especially in detecting a single poison, or in estimating it quantitatively. For instance, pure ether would not be the best solvent to use in extracting strychnine quantitatively from an alkaline solution. A mixture of ether and chloroform, or better pure chloroform would be preferable, since strychnine is more soluble in the latter solvent than in pure ether. For the same reason chloroform should be used in the quantitative extraction of caffeine or antipyrine. When only a small quantity of material is available for analysis, tests for all three groups of poisons may be made with the same portion. In this case after removal of volatile poisons the residue should be divided into two unequal portions. The larger portion should be tested for non-volatile, organic poisons. The smaller portion, together with the residue left after extracting non-volatile, organic poisons should be tested for poisonous metals. It is advisable, however, even in such a case to reserve a portion of material for any contingencies.

Organs of the human body like liver, kidneys, spleen, heart, brain, stomach, or intestines with contents, should be cut into small pieces and then finely chopped before being examined chemically. An organ should first be cut into small pieces with sharp, clean scissors and then minced with a clean chopping knife in a new wooden bowl, or a small meat machine, which has been carefully cleaned, may be used. Material may be held with nickel plated tongs while being cut with scissors.

Marsh's Test for Arsenic.—When hydrogen is generated in the presence of compounds of arsenic, they give up the arsenic, which, uniting with hydrogen, forms arsenic terhydrid, AsH_3 . This is a gas which, by heat, yields the metallic arsenic for identification by tests already stated. In a flask arranged for generating hydrogen, with air-

tight connections, pure zinc is placed, and pure cold dilute sulphuric acid is added to it through the funnel tube. The gas is first conducted through a drying tube containing calcium chlorid between plugs of glass wool, and then through an exit tube of hard glass, about 5 mm. internal diameter, and 25 to 50 cm. long, which is turned up at the end and drawn out at the tip to make a jet. After waiting a few minutes for the air in the apparatus to escape a Bunsen flame is applied in the course of the exit tube, which is heated to a low red heat, and if no stain appears on the glass after fifteen minutes, the chemicals may be considered pure. The gas jet should be ignited, and if arsenic fluid is now poured in by the funnel tube in small portions, the pale hydrogen jet becomes more luminous and livid in color. If organic matter should cause much frothing, a small quantity of alcohol may be introduced by the funnel tube.

Fallacies.—Antimony is deposited under the same conditions as arsenic, and in a form closely resembling it, whether in the spots on porcelain or the mirror-like ring in the heated tube, but the arsenic mirror is at a little distance beyond the flame and brownish shading to black, while the antimony is close to the flame, sometimes on both sides of it, and tin-like in luster. (Holland Medical Chemistry and Toxicology.)

SIMPLE CHARACTERISTIC TESTS FOR SOME IMPORTANT POISONS.

Inorganic:

Acids.—Turn blue litmus paper red; dissolve zinc with liberation of hydrogen gas.

Hydrochloric.—With silver nitrate, white curdy precipitate which turns blue to black on exposure to light.

Sulphuric.—With barium chlorid solution, fine white crystalline precipitate insoluble in strong HCl.

Nitric.—Liberates nitric oxid fumes when acting on copper or a silver coin.

Alkalies.—Turn red litmus paper blue; solutions feel soapy when rubbed between the fingers.

Antimony compounds.—In acid solution give an orange colored precipitate with hydrogen sulphid.

Arsenic compounds.—In acid solution give a yellow colored precipitate with hydrogen sulphid.

Barium compounds.—In acid solution give a fine white precipitate with sulphuric acid or a solution of a sulphate.

Copper compounds.—Solutions turned an intense blue upon addition of an excess of ammonium hydroxid; give a chocolate brown precipitate with solutions of potassium ferro-cyanid.

Iodin.—Turns starch paste an intense blue.

Lead compounds.—Solutions give a brilliant yellow precipitate with potassium chromate, a white precipitate with strong sodium sulphate solution, and a black precipitate with hydrogen sulphid.

Mercuric compounds.—Scarlet precipitate with potassium iodid solution, which dissolves in excess of reagent; white precipitate with ammonium hydroxid.

Phosphorus.—Displays phosphorescence in the dark; oxidized surface of mass dissolves in water and gives acid reaction, tastes sour and gives white precipitate with magnesia mixture.

Silver compounds.—Give with solution of a soluble chlorid a white curdy precipitate, which changes in the light to a blue-black.

Organic:

Alkaloids.—In clear solution give a yellow precipitate with alcoholic solution of picric acid.

Atropin.—Dissolved in sulphuric acid and warmed gives odor of rose perfume; a few grains of potassium bichromate added to this cause odor of bitter almonds and change color to green.

Cocain.—Solution treated with concentrated aqueous solution of sodium nitro-prusside, gives turbidity due to formation of microscopic reddish crystals that dissolve on warming.

Morphin.—Nitric acid dropped on a particle of solid gives blood-red solution, changing gradually to orange-yellow.

Strychnin.—Dissolved in sulphuric acid gives rise to no color; draw a crystal of potassium bichromate thru the solution and a play of colors takes place, —blue, purple, cherry, pink, yellow.

Cantharides.—Recognized by the fluorescent green particles of the insect.

Formaldehyde.—Can be recognized by odor; or add a little to milk and apply sulphuric-acid-ferric-chlorid test.

Hydrocyanic acid.—Odor of bitter almonds; add to solution some potassium hydroxid, then a mixed solution of ferric and ferrous sulphates will give a dirty green-blue precipitate which turns to bright blue on heating with hydrochloric acid.

Oxalic acid.—Crystals converted to invisible gas on heating; solutions give white precipitate with lime-water.

POISONING BY SNAKE VENOM.

Altho there are many different varieties of poisonous serpents and other reptiles, and the symptoms following their bites differ both in intensity and character, the general mode of treatment of such cases of poison is always the same. In this outline we shall confine our description of symptoms to those produced by the venoms of the *Crotalus* family, which is the most common of the venomous families in America. For a thoro discussion of the subject of snakes and their venoms the reader is referred to FitzSimons, "The Snakes of South Africa: Their Venom and the Treatment of Snake Bite;" and, Noguchi, "Snake Venoms," published by the Carnegie Institution of Washington.

The action of the venoms of the rattlesnake, copperhead and water-moccasin is due predominantly to the presence of toxin called hemorrhagin, and to a much less degree to an agglutinin, hemolysin and neurotoxin, this latter being the most important in cobra venom. The effect of the hemorrhagin is to so damage the endothelial linings of all blood and lymph vessels reached by the venom that they are incapable of preventing extravassation of the blood and lymph. The local effect, if the amount of venom injected be large, may be so profound as to induce gangrene; the systemic effect and death are largely due to the capillary hemorrhages into the vital nerve centers, resulting in their loss of ability to control the vital functions of the body. The picture presented by a case of poisoning will usually be complicated by shock due to fright and in no manner dependent upon the action of the toxin upon the central nervous system.

One property of the venoms demands especial emphasis. They are of the nature of true toxins and therefore act as antigens, exciting the generation in the animal body of specific antivenoms capable of completely neutralizing their poisonous action. This ability of the higher animals to develop antivenoms, however, exerts itself too slowly to be of value in protecting the animal from a single large injection of venom. Repeated small injections (whether artificial or by repeated

small snake bites) develop an active immunity in the animal. This fact is utilized in the preparation of antivenoms, for use in snake bite, in a manner entirely similar to the preparation and use of diphtheria antitoxin. Such antivenoms are usually obtainable at sporting goods stores and at some drug stores. To be of value they must be used promptly as indicated in the discussion of the treatment below.

The degree of danger from a snake bite with any given variety, will depend upon the amount of venom injected and the degree to which its action is confined to local tissues or permitted by absorption to extend itself to the general system. The larger the snake, the greater the quantity of venom, as a rule. The deeper the fang wound, the greater the likelihood of absorption. Prompt and adequate treatment will usually save victims of snake bite who otherwise would be almost certainly fatally stricken. It is impossible to make any statement of the mortality following snake bite, even when the variety of snake is known. "The average mortality from the bite of the more common American venomous snakes is extremely low."

Following the bite of a snake **the local symptoms are most pronounced at first**. The wound itself is usually painful and often bleeds rather freely. Very soon the tissue around the wound becomes swollen and discolored, and as the action of the venom extends up the limb the swelling spreads and is soon followed by discoloration suggestive of a bruise. Pain accompanies the swelling of the part. In extreme cases, the swelling of the limb is followed by the appearance of vesicles upon its surface; the lowering of the temperature of the part as the circulation becomes defective, and loss of painful sensation in the part follow. Finally gangrene may result. If there is rapid subsidence of the local symptoms, it is probably due to the fact that the amount of venom injected was small and has expended its virulence, not due to the use of some reputed "cure."

Prompt development of **systemic symptoms** is due often rather to the prostration from fright than from the central action of the venom. There are, however, true cases of prostration resulting from the rapid absorption of the venom and its action upon the central nervous system. These early symptoms are those of shock,—weakness, loss of power of locomotion introduced by staggering and falling, cold sweat, anxious face, difficult respiration, vomiting (sometimes diarrhea), and small, rapid pulse.

There may be gradual but widely spreading development of the symptoms. The swelling and discoloration instead of being so sharply localized as to result in gangrene, may extend itself so as to include the trunk and cause puffiness of the

face. The systemic symptoms instead of displaying a typical shock picture may show persistent weakness with dyspnea and tendency toward syncope. Restlessness, insomnia and delirium or merely a more moderate disturbance of cerebral activities may be experienced. In fatal cases death may occur anytime between six hours and several days, seldom in less than six hours unless the shock from fright be a dominant factor.

For the adequate treatment there must be available for immediate employment:

A ligature,—a strong elastic band or a strip of heavy cloth.

A knife for incising area about the wound.

Potassium permanganate, crystals, or preferably in the form of a readily soluble hypodermic tablet.

A supply of specific antivenom and hypodermic syringe for administering it.

Promptness of treatment is most urgent. Five minutes after the bite is received the potassium permanganate will be of no value and three minutes will often be too late for its value to be great. One or two minutes' delay in applying the ligature may permit the case to terminate fatally as the result of the absorption of the venom.

Proceed as follows:

(1) **Instantly after the bite has been made, apply the ligature** firmly, so as to check return of venous blood and lymph, placing it just above the knee or just above the elbow, according as the bite is on the leg or the forearm. If any delay is met in applying the ligature, so that it cannot be gotten on in less than a minute or at most two minutes, proceed without waiting to (2) and (3).

(2) **Make several cuts at and about the location of the bite.** Excepting when the bite is so located that there is danger of cutting ligaments or large blood vessels, make the incisions criss-cross and as deep, at least, as the fang marks. Over the fleshy parts of the hand or the calf of the leg the incisions may be made $\frac{1}{4}$ to $\frac{1}{2}$ inch deep, even going to the bone.

(3) **Crush a few potassium permanganate tablets in the palm of the hand and dissolve in some saliva or a little water if it is immediately at hand. Rub and press this strong permanganate solution thoroly into the wounds.** If possible also inject some permanganate solution (1 grain to a teaspoon of water), by means of a hypodermic syringe into the depths of the fang wound and into the tissues immediately surrounding.

Remember that all of the preceding steps should have been taken within three minutes from the moment of the bite. If

anyone is available to help, the ligaturing and scarifying and treatment with permanganate may be going on at the same time.

(4) **Inject the antivenom serum hypodermically in several places** upon the limb, near the wound but outside of the area of action of the permanganate solution, as well as at places remote from the wound.

If true systemic symptoms are appearing give an intravenous injection also. As much as 50 or 100 c.c. of the serum may be used in severe cases. In animal experimentation the injection of antivenom will frequently save the lives of moribund animals.

(5) "Get the patient to bed, wrap him up well and apply hot-water bottles, jars, or hot fomentations to the feet, abdomen, armpits, and back of neck, if he is in a serious state of collapse. It is a mistake to walk the patient about with the object of keeping him awake."

(6) "Be careful to see that a ligature does not remain on a limb for longer than half an hour, or an hour at most, otherwise there is grave danger of the part mortifying. It is not desirable to remove the ligature at once. It should be gradually slackened over a period of an hour or several hours and eventually removed. The object of this is to prevent the venom getting into the general circulation all at once."

Systemic treatment aside from managing the collapse or shock, after the administration of antivenom, is usually superfluous and often injurious. "It is a grave error to dose a patient with alcohol or ammonia." (FitzSimons.) In animal experimentation by Mitchell and Reichert it was found that alcohol aggravated the symptoms and hastened death. In a study of a series of 426 cases of snake bite in humans, Huxtable found the mortality when strychnine was used in the treatment to be 18.2% ; where strychnin was not used the mortality was only 2.4%. As in the case of shock and collapse from other causes, it seems that following the usual medicinal methods of treatment results simply in the unnecessary killing of a rather considerable number of patients by the stupid use of the irrational drugs.

DIFFERENTIAL DIAGNOSIS IN THE MOST COMMON FORMS OF COMA.

Uremic.—Odor: Urinous.

Pulse: Rapid and high-tension.

Respiration: Dyspnea and irregular.

Pupils: Contracted; may react to light.

Extremities: Rigid and legs are edematous.

Temperature: Usually elevated.

Face: Dusky countenance and edema.

Urine: Contains albumin.

Apoplectic.—Pulse: Slow and full.

Respiration: Deep, slow, noisy and stertorous.

Pupils: Dilated and unequal. Do not react.

There is conjugate deviation.

Hemiplegia: Paralyzed leg is flaccid, other leg rigid.

Extremities are cold.

Temperature: At first subnormal.

Face: Ashen-grey or cyanotic.

Coma complete. Cannot be aroused.

Alcoholic.—Odor: Alcoholic.

Pulse: Full and fairly normal.

Respiration: Decreased but comfortable.

Pupils: Dilated and fixed.

Temperature: Subnormal.

Face: Flushed.

Coma: Not deep; talks irrationally.

Complete relaxation.

Opium.—Odor: of opium.

Pulse: Slow, forcible and very good.

Respiration: Greatly reduced; 2 to 12 per minute.

Pupils: Pin-point; do not react to light.

Face: Cyanosis, profuse sweat, and warm.

Coma: Not deep; answers questions, but goes to sleep between them.

Epileptic.—Pupils: Dilated and insensitive.

Epigastric auræ.

History of cry when falling.

Biting of tongue due to spasm of the jaw.

Frothy and bloody expectoration.

Face: Pale and turned to one side and fixed.

Coma is of short duration.

Compression of the Brain.—Bradycardia.

Respiration: Stertorous and Cheyne-Stokes.

Pupils: Dilated and insensitive. Choked disc.

Temperature present and skin is flushed.

Sphincters paralyzed.

Paralysis: First spastic, then relaxation of the extremities occurs.

(Merck's Manual, 4th Ed.)

PHARMACOLOGY STUDY QUESTIONS.

(Concerning Irritant and Inorganic Drugs.)

Based on Green's Pharmacology.

1. What is meant by an irritant drug? by a corrosive? (XXXIII.)
2. Distinguish between the meanings of the terms irritant and stimulus.
3. Contrast the response of a tissue to a normal stimulus and to an irritant.
4. Why is even excessive stimulation of normal nature less likely to cause degeneration of a functioning tissue than is irritant stimulation?
5. Which cells of an organ usually are first affected by irritants?
6. Describe four different degrees in the effects of irritants.
7. Why in excretory organs do the functional cells show much greater effects of the action of irritants than do the interstitial cells?
8. Give a probable explanation for the fact that all tissues and cells are not equally affected by any given irritant or other drug, and illustrate answer with description of reaction of cells to stains.
9. Why are most drugs relatively inactive when applied to the skin? (XXXIV.)
10. What two properties are possessed by the drugs in use as skin-irritants?
11. Explain the value of an inflammatory reaction to the injury produced by a skin irritant.
12. What is a counter-irritant effect? How are such reactions produced? Explain nerve mechanism involved. (XXXVI.)
13. Name four skin irritant drugs and state their chemical nature.
14. What are the effects—systemic—after absorption of members of this group? Explain why a mustard plaster should not be made up with hot water, and why its action becomes more intense the longer it acts.
15. What functional change in the central nervous system is set up by all peripheral irritations when of mild degree? When excessive? (XXXVI.)
16. What is the general systemic response to mild but extensive application of cutaneous counter irritant influences?

17. Into what three chemical groups are the vegetable cathartics (XXXV) divided? Group the following according to chemical nature of active principle: croton oil, podophyllum, jalap, castor oil, rhubarb, cascara, elaterium, aloes.

18. To which group is phenolphthalein related?

19. Why are all of the vegetable cathartics practically inactive in the stomach?

20. What is the most common effect of many of these drugs if they are absorbed?

21. Why are members of this group usually not absorbed?

22. Do they ever produce systemic effects without being absorbed and if so, what? How?

23. Upon what local effect does the cathartic influence depend?

24. What influence have members of this group upon the three functions of the intestines,—secretion, absorption, motility; and what part does each of these influences have upon evacuation of the contents of the bowels?

25. What is the evidence that, under ordinary circumstances, the fluidity of the intestinal contents is not due to an inflammatory exudate?

26. Why is extreme inflammation rarely excited by even the more drastic purgative drugs? Under what circumstances may this result ensue?

27. Explain the apparent cholagogue action of the members of this group.

28. Why are they contraindicated (excepting the mildest members) in cases of nephritis?

29. How long a time is required for the cathartic action to appear following therapeutic dose of croton oil, elaterium, jalap, cascara, senna, podophyllum, castor oil, phenolphthalein?

30. What evidence indicates that phenolphthalein as therapeutically used is not entirely harmless?

31. Explain the prolonged cathartic action of phenolphthalein.

32. What chemical change takes place in the cathartic oils before their action is developed?

33. Which intestinal function is particularly augmented by the anthracene relatives?

34. Name three members of the group which are apt to set up intestinal inflammation.

35. Would castor oil in an enema specifically accelerate evacuation?

36. Define—crystalloid, colloid, dissociation, cation, anion, ion, electrolyte, solution, diffusion, osmosis, osmotic pressure, permeable membrane, semi-permeable membrane, non-permeable membrane, isotonic solution, hypertonic sol., hypotonic sol., salt action. (XXXVII.)

37. Compare a tissue cell to an osmotic apparatus, and describe how the permeability of its surface influences its metabolism.

38. What is the effect of direct exposure of tissue cells to pure water (a) structurally, (b) functionally? (XXXVIII.)

39. Explain the fact that drinking large quantities of water fails to notably dilute the body fluids.

40. Explain the effects of drinking large quantities of water (a) upon metabolism and (b) upon elimination.

41. What is the composition of physiological saline solution? of Ringer's solution? of Locke's solution? (XXXIX.)

42. Why is physiologic saline solution in contact with tissue preferable to distilled water? Why is it unsuited for the maintenance of vitality in a tissue when the contact is to be prolonged?

43. What is the object of the injection of saline solutions after hemorrhage? during severe toxemia?

44. Why is a saline solution for injection preferable to plain water?

45. Why is Ringer's solution preferable to physiological saline?

46. What is the effect on a cell of exposure to a solution containing a predominance of sodium chlorid as compared with K and Ca salts? (XL.)

47. Why is the use of large quantities of sodium chlorid in the diet not followed by more distinct influence upon metabolism?

48. Why is the therapeutic use of bromid or iodid of sodium preferable usually to the use of the corresponding potassium salt?

49. Concerning the bromides, what is their most specific effect, their influence on the skin after prolonged administration, and their influence upon the gastro-intestinal tract?

50. What organ appears to be stimulated by the iodides? What is the effect of iodides upon normal and excessive blood-pressures? Explain.

51. What is the effect of prolonged use of iodides upon the skin and mucous membranes? Their effect upon active hyperplasias?

52. What tissue is particularly stimulated by the nitrates after their absorption?

53. Why are the absorbable salts of sodium required in large frequent doses to produce a systematic effect?

54. What is the effect of the sulfate ion on muscular tissue?

55. Contrast the effect of potassium and calcium ion influence upon muscle irritability, contractility, and ability to relax, when these ion concentrations are separately increased. (XLI.)

56. Why is the potassium influence rarely felt after administration of its salts by mouth? What is potassium influence on nerve tissue?

57. Explain the expectorant action of ammonium chlorid. (XL.)

58. What is the difference in the mode of elimination of ammonium chloride and ammonium carbonate or acetate?

59. What is the effect of increased concentration of ammonium ion in the circulating body fluids? Deficiency of what organs (3) seems to favor this condition?

60. What are the direct and reflex effects of exposure of the respiratory mucous membrane to ammonia vapor?

61. What popular supposed actions of lithium have been proven absent? (XLI.)

62. State the relation of calcium in the body fluids to blood-coagulation, to permeability of cell membranes, to muscle contraction and irritability and relaxation, to motor nerve ending function, to central nervous system tissue, telling so far as possible effect of excessive and deficient concentrations.

63. Is the calcium in the tissues present only in inorganic form?

64. By what two channels is calcium excreted?

65. Why does not eating fruits containing citrates interfere with the coagulability of the blood?

66. Give two reasons for the lack of success in many cases to raise the coagulability of the blood by administration of calcium salts.

67. What is the most important magnesium action in the body?

68. Name four of the Saline Cathartics. (XLII.)

69. What observations made by Hertz indicate the error of believing that the fluid contained in the stool following the administration is derived by osmotic abstraction from the walls of the intestines?

70. What observations indicate the error of believing that the drugs considered as saline cathartics are not absorbed from the intestines?

71. What change in the colon following the administration of a saline cathartic is directly responsible for the initiation of defecation?

72. What is the influence of a strong solution of one of the salines upon the stomach? How may this influence effect the activity of the lower bowel so as to hasten defecation?

73. What portion of the alimentary tract is least affected by the administration of an ordinary dose of a saline? Most affected?

74. Which of the ions frequently introduced in the salines has the most marked effect upon the musculature of the intestines after absorption?

75. Explain why the magnesium ion is largely excreted thru the feces.

76. Give two reasons for administering the salines with a large quantity of water.

77. What is the influence of the salines upon absorption from the intestines, and how does this influence intestinal movements? Are the effects due to the direct action of the drug on the mucous membrane or are they caused after absorption?

78. What is the influence of the salines upon the kidneys?

79. Under what condition do the salines remove fluid from the body best?

80. Why are the salines not reliable cleansers of the whole intestines?

81. State two manners in which the salines in enemas increase their effect.

82. Which ions are responsible for acid and alkaline reaction, respectively? (XLIII.)

83. To what is the normal alkalinity of the tissues due?

84. Why is the maintenance of a normal degree of alkalinity in the body of great importance? How would sodium citrate taken by mouth influence the reaction of urine?

85. Contrast the effect of a strong alkali and a strong mineral acid when applied to the skin.

86. Why is sodium carbonate less safe than sodium bicarbonate as an alkalizing agent?

87. What are the reflex effects of the action of strong acids on the mucous membranes of the digestive tract?

88. What is the difference between the fate of the organic and the inorganic acids after absorption? Are all organic acids disposed of in the same manner? Which organic acids acidify the urine?

89. What is meant by acidosis and what is the value of the administration of alkalies in this condition?

90. What is the effect upon metabolism of the withdrawal of oxygen? (XLIV.)

91. What is the effect upon nerve tissue of a moderate diminution of the oxygen in the fluids circulating thru it?

92. Why, under normal conditions, does it not accelerate metabolism when oxygen is breathed instead of air? Under what conditions may the breathing of oxygen be of some real assistance to the body?

93. In shock what is the relation of the carbon dioxide of the blood to venous stasis?

94. What is the effect of acidosis upon the ability of the blood to carry carbon dioxide away from the tissues?

95. What is the action of hydrogen peroxide when placed in contact with the mucous membranes? Of potassium permanganate?

96. Regarding the following, state which are true metals, which metalloids, (XLV), which are non-metals, which form salts that precipitate albumin, which do not precipitate albumin, which are important as nutrients, which readily absorbed, which difficultly absorbed:—Copper, lead, mercury, iron, zinc, silver, arsenic, phosphorus, antimony, sulphur, bismuth.

97. Why are organic salts of the metals less intense in their action than inorganic salts? (Two reasons.)

98. Compare the actions of zinc stearate, acetate, sulphate, and chlorid and explain the differences in their actions.

99. What is the influence of an excess of albumin upon a metal albuminate? Of sodium chlorid solution?

100. Why do compounds of the heavy metals produce systemic symptoms in only a few cases?

101. Give evidence proving that inorganic iron can be absorbed. (XLVI.)

102. Under what conditions and in what manner does iron act definitely as an hematinic when administered in inorganic form?

103. What detrimental effects are apt to accompany such use of iron?

104. What advantage is possessed by the organic preparations of iron?

105. By what channel is iron largely excreted?

106. In the alimentary tract what is the effect of sulphur? What compound formed from the sulphur is the cause of this action? (XLVII.)

107. In what form does absorption take place when sulphur is administered by mouth? What is the predominant action of this form in the system?

108. Explain the parasiticial action of sulphur on the skin.

109. What is the local effect of phosphorus?

110. Describe the effects of phosphorus upon the structure of bone, during the early and late stages of its administration. Explain cause.

111. What are its effects upon the tissues of the body at large after absorption? Upon amount and form of nitrogen in urine in early stages?

112. Why does phosphorus produce, when used in very small doses, an increase of erythrocytes followed by a diminution in their number?

113. What are the functions of inorganic phosphates in the diet?

114. What are the three forms of organic phosphorus compounds recognized?

115. State the differences between the effects of phospho-proteins and nucleo-proteins upon the body. (XLVIII.)

116. How does the local injury caused by arsenic differ from that caused by most corrosives? (XLIX.)

117. Explain the origin of "rice water stools" following large doses of arsenic. Explain cause of edema in early stage of chronic poisoning.

118. Contrast the hematinic actions of iron and arsenic compounds.

119. In minute doses what is the primary effect of arsenic upon the tissues of the whole body? Effect of prolonged or excessive dosage?

120. Name four important organic compounds of arsenic used in therapeutics. Why are these compounds much less toxic than an equivalent amount of arsenic?

121. What is the effect of arsenic in the blood upon the arteriole walls and upon heart muscle?

122. Compare actions of antimony and arsenic. Why is antimony less available for its systemic effects than arsenic?

123. State the manner in which each of the following conditions is caused by the action of lead after absorption:—"lead colic," "lead line" on the gums or teeth, constipation, wrist drop, nephritis. (L.)

124. Why does lead continue to be excreted for so long a time after its administration has been discontinued?

125. What is effect of lead upon the relaxation of muscle tissue?

126. Why is zinc less absorbable than lead? (LI.)

127. Contrast the actions of ZnO and ZnCl_2 upon the skin.

128. Contrast the effects of lead and copper after absorption. What is the effect of repeated action of copper present as an impurity in food? (LII.)

129. What is the difference between the effect of silver nitrate and the soluble organic silver compounds upon albumins and the chlorids? Which is the more strongly antiseptic?

130. What is the fate of silver after absorption? (LIV.)

131. Why is it not entirely safe to use bismuth compounds for prolonged application to ulcerating surfaces? Why may large doses of bismuth sub-carbonate be given safely in Roentgenographic work? Why is the subcarbonate preferable to the subnitrate? What is the general influence of these compounds upon the gastro-intestinal tract? (LV.)

132. Why are mercury compounds so highly toxic compared with other metallic compounds? (LIII.) Why calomel permissible in relatively large doses?

133. Primary effect of minute doses of mercuric chlorid systematically? Effect of long continued use of mercury?

134. Mode of action and site of action in production of catharsis? Effect upon kidney secretion and kidney cells?

135. By what channels is mercury excreted and what are the symptoms arising in this connection?

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